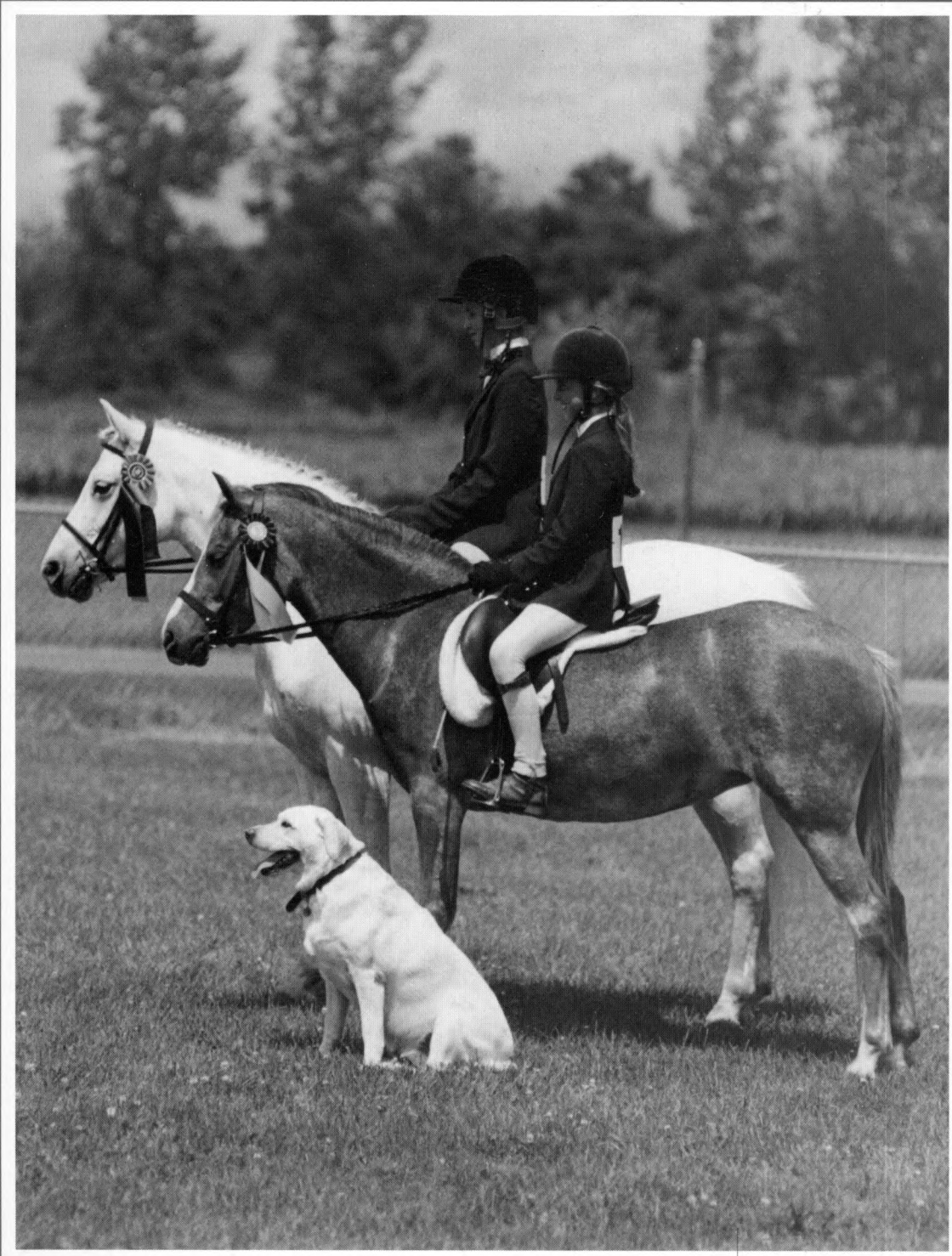


JAMES A. BAKER INSTITUTE FOR ANIMAL HEALTH

Annual Report 1992



College of Veterinary Medicine, Cornell University

VOLUME 42

Cover: GlanNant Serenade and GlanNant Strawberry Fields (foreground), registered Welsh Ponies owned by the late Mrs. Karl D. Butler of GlanNant Farm, Ithaca, New York. The ponies were ridden and shown in 1992 by Lizzie and Harriet Antczak, who are pictured with their Labrador Retriever, Maisie.

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ALEXIS WENSKI-ROBERTS

Harriet, Maisie and Lizzie Antczak

A Message from the Director

This year has witnessed extraordinary growth in the Baker Institute's faculty and programs as well as substantial expansion and refurbishment of the facilities that accommodate the research enterprise. The year has been one of the most productive and satisfying in our history. The progress reported in the pages that follow could not have been achieved without the generous financial support of our benefactors, and it is primarily for them that we prepare our annual report. I am pleased again to acknowledge the many dog owners, kennel clubs, foundations, veterinarians in practice, firms engaged in private enterprise, and individuals who have recognized the importance to animal health of the Institute's research over the past 42 years. I also want to take this opportunity to express my gratitude to the Institute faculty and staff for their tremendous support during my interim term as director, and to Dean Robert Phemister, whose help and advice have been greatly appreciated. It has been my special privilege to work with all of them during the transition.

We are entering a new era in the life of the Institute as we welcome the arrival of **Gustavo Aguirre** to succeed me as director in the spring of 1993. Dr. Aguirre, a distinguished comparative ophthalmologist and geneticist with proven success as a research scientist and administrator, emerged from a field of fine candidates as an exceptional match for the position. He has already completed the movement of his laboratory and staff from the School of Veterinary Medicine of the University of Pennsylvania and established his research programs in canine genetics at the Institute. Together with another Institute newcomer, **Vicki Meyers-Wallen**, and colleagues **Gregory Acland**, **Jharna Ray**, and **Bennett Hershfield**, Dr. Aguirre has established the Center for Canine Genetics and Reproduction. These new faculty members have increased the fund of talent that will endow many of Dr. Aguirre's undertakings as director of the Institute. We all look forward to a

long and productive association.

Our previous director, Associate Dean for Research Douglas McGregor, launched the Institute into the modern scientific era with the introduction of molecular biology and new faculty trained in the basic sciences, especially genetics, virology, and immunology. With strategies for the control of the major dog diseases now largely successful, our research has expanded in the past decade to include studies of reproductive biology, especially of the dog and horse, and the genetic basis of hip dysplasia. Our traditional studies of infectious diseases continue to occupy a substantial portion of the Institute's activities, although the problems are different. Future research at the Institute will place even more emphasis on canine and equine genetics. Gus Aguirre's group studies progressive retinal atrophy and other inherited eye diseases of dogs. **James MacLeod** has established new programs examining molecular and cellular regulators of growth and development in bones and joints. Dr. Meyers-Wallen's research focuses on inherited reproductive diseases of dogs. With strong input from the faculty and our Advisory Council, we have been able to reach consensus on our future research goals and have made a substantial investment during the past year in the refurbishment of laboratories and graduate student facilities, the enhancement of computer capabilities, and the construction of laboratories to accommodate the new scientists.

At the annual meeting of our Advisory Council, we take the opportunity to honor individuals who have made especially significant contributions to the success of the Institute. For the past eleven years we have, on that occasion, presented the **Arthur F. North, Jr. Canine Service Award** to an outstanding champion of canine health and welfare. This year we had the great pleasure of recognizing **Eleanor S. Gillis**, a friend of many years, for her dedication to dogs and to furthering Institute research for their benefit. Eleanor and her late husband,

Gordon, made provisions in their wills to establish an endowment at the Institute that will, when fully funded, promote the work of our most promising young investigators.

The Founders' Award recognizes exceptional contributions to the veterinary profession and to the advancement of canine health research. It was presented this year to **Niel Pieper**, who not only has served the veterinary profession with extraordinary distinction, but has been a good friend of the Institute for decades. At that meeting we also unveiled a portrait of James A. Baker, the Institute's founding director, which was sponsored by his many friends. It was painted from a series of photographs taken at various times and attempted to capture his individuality as well as his physical features.

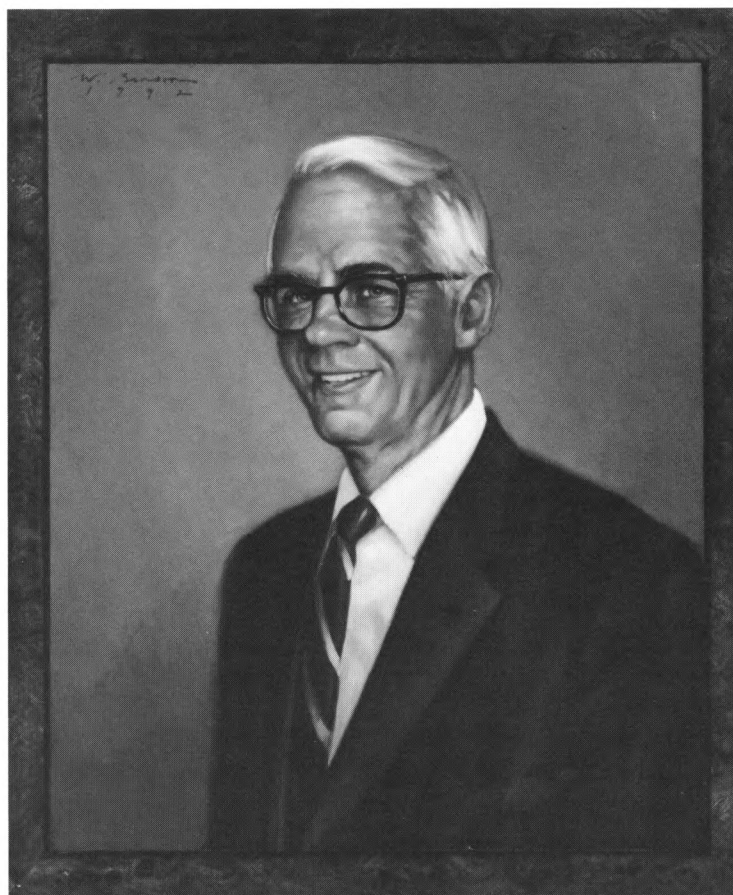
We were greatly saddened over the death last April of Gaylord Donnelley. He and Dorothy, his wife, have been special friends for many years, and their generosity and wisdom have influenced the course of Institute research since our founding. Gaylord Donnelley lived a life of remarkable accomplishment as a businessman and philanthropist and will be remembered for his enormous contributions to environmental conservation and the health and welfare of working dogs and wildlife. His loss is deeply felt by the great many people who had the privilege of knowing him. Doug McGregor and I were fortunate to visit with him and Dorothy at his beloved Ashpoo Plantation on the eve of his death, and he will endure in our memory.

Before closing, I wish to thank our Advisory Council for the commitment and support they have provided in guiding the Institute. My colleagues and I are especially grateful for the efforts of Robert Shope, the chairman of our Advisory Council. I am sure that Gus Aguirre will derive as much pleasure as I have from working with such an exceptionally dedicated and talented group of individuals.

Leland E. Carmichael

James A. Baker Portrait

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William Benson 1992

The funds for Dr. Baker's portrait were provided
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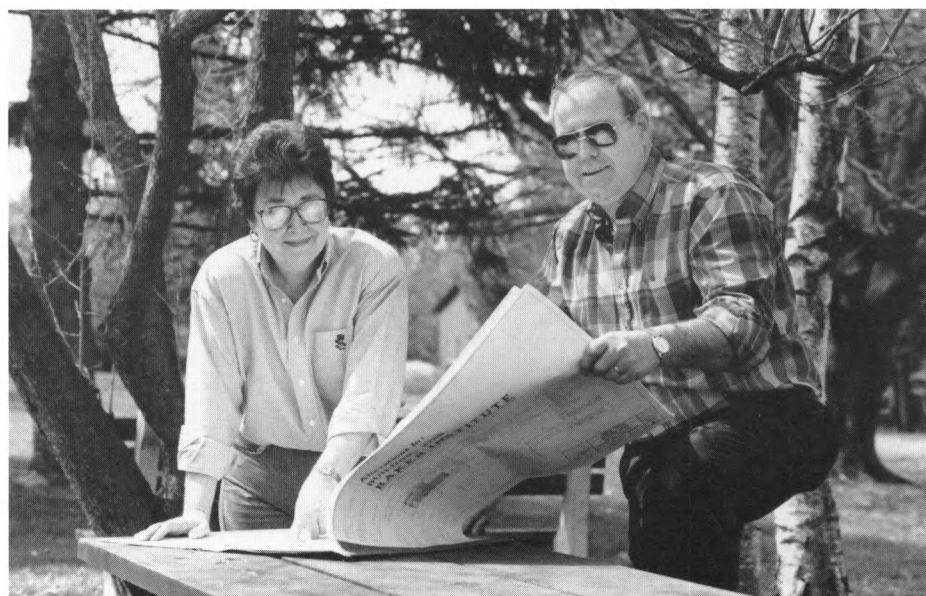
Custodian

RICHARD E. DANIELS

Maintenance mechanic

RUSSELL F. HAUS

Maintenance mechanic



Susan Hamlin and Arthur Howser

DEDE HATCH

Giralda Laboratory for Canine Infectious Diseases

Canine Parvovirus Vaccines

During the past three years many individuals have inquired whether new canine parvovirus (CPV) vaccines are needed, since some vaccinated dogs have subsequently succumbed to the disease. Studies over the past several years do not suggest a need for new vaccines. They do reveal, however, that certain vaccines have not always maintained sustained efficacy. More importantly, they show that owners do not fully perceive the need for strict hygiene during the period from birth to four months when residual maternal antibodies may interfere with a vaccine's ability to immunize a pup. We are encouraged that CPV-2 vaccines have generally been efficacious: a recent survey of the immune status of nearly 1000 breeding-age dogs revealed an immunity level greater than 87%.

The question of whether new CPV-2 vaccines were needed was prompted by Colin Parrish's discovery that at least three distinct antigenic types of this virus have emerged in the dog population since the virus was first recognized in 1978. The antigenic types were initially defined by comparing the serological profiles of viral isolates collected from several areas of the world over a period of about eight years following the appearance of CPV-2. Dr. Parrish also observed similar differences among isolates of the closely related mink and feline parvoviruses. Such variation seems to be a normal consequence of parvovirus evolution and diversity over time.

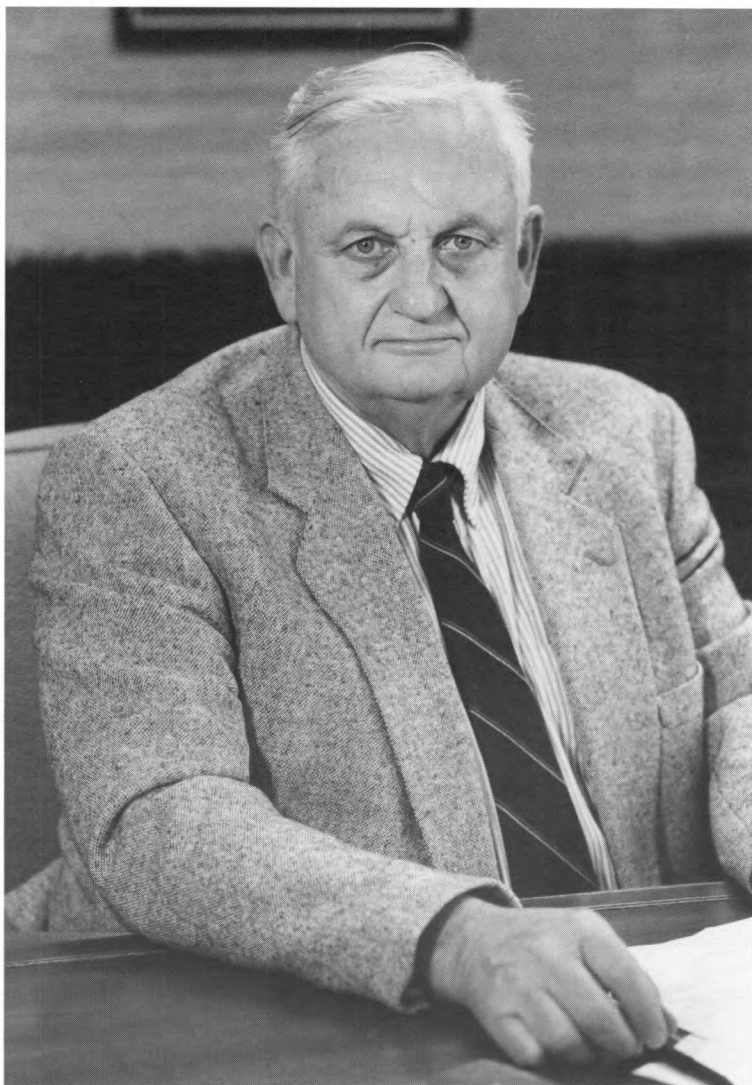
The most recent variant recognized by Dr. Parrish is CPV-2b, a virus type that appeared around 1984. As a consequence of the finding that most CPV-2 strains currently circulating in the dog population differ from the 1978 virus that constitutes most vaccines, the misconception has grown that new CPV vaccines are needed. New

vaccines may indeed provide benefit, but not for the reasons commonly assumed. As explained in earlier reports, CPV-2 vaccines may differ in efficacy according to the extent to which they have been cultivated in the laboratory.

Studies over the past 12 years have revealed that virtually all effective, commercially licensed CPV-2 vaccines do protect against the new antigenic types. However, some vaccines have suffered at one time or another from inadequate potency—the ability to immunize dogs—when non-

immunizing mutants emerged in cell cultures during vaccine production. As a consequence, several "second-generation" CPV-2 vaccines have appeared as commercial products.

An attenuated strain of the currently prevalent CPV-2b has been developed at the Institute in order to study whether it possesses advantages over the previous types. As noted further on in Dr. Parrish's report, Allen Gruenberg has now succeeded in cloning the entire genome of the attenuated CPV-2b into a bacterial plasmid. The cloned virus genome,



Leland Carmichael

CHARLES HARRINGTON

inserted into a common species of bacteria, *E. coli*, can be quickly amplified for the purposes of providing large amounts of DNA to study fundamental viral properties; it also allows the preservation of stable vaccine stock. Although we have not been able to demonstrate that the CPV-2b vaccine offers any significant advantage over the original CPV strains in immunizing pups, we have determined that it elicits vigorous immune responses and is safe. An important feature of the cloned virus stock is that it retained its immunogenicity for dogs after several passages in cell culture, a trait that should decrease the frequency of emergence of non-immunizing, mutant virus during vaccine production.

A brochure that explains the current vaccination situation and provides comments and guidelines on vaccination strategies, *Canine Parvovirus Vaccination*, is available from the Institute without charge to those who request it.

Canine Parvovirus Pathogenesis

David Peters, a National Institutes of Health DVM Postdoctoral Trainee, joined our laboratory last year to pursue graduate studies after completing a residency in the Department of Pathology. Dr. Peters is studying the pathogenesis of CPV-2b to determine ways that the new variant might differ from the original CPV-2 strain in its mode of growth. The goal of his studies is to characterize the mechanisms by which CPV-2b infects dogs and provokes disease as well as to examine differences in dogs' responses to the derivative vaccinal strain. His studies would augment the genetic differences in the two viruses that have been revealed in the molecular studies of Dr. Parrish and Dr. Gruenberg.

Minute Virus of Canines

We continue to study the role of "minute virus of canines" (MVC) in natural disease. MVC, which is also known as CPV-1, is distinct from the more familiar CPV-2. MVC may cause fetal resorptions or abortions in pregnant dams exposed during early gestation and a fatal illness, characterized by viral pneumonia and enteritis, in very young pups. Thirteen fatal cases of MVC were reported last year. Most of those pups had died at ages between one and three weeks and all had severe enteritis similar to that caused by CPV-2. In contrast to CPV-2, however, evidence of MVC viral growth (antigen and inclusion bodies) predominated in the body and tips of intestinal villi rather than in cryptal cells, as is characteristic of CPV-2. Additional cases of MVC have been diagnosed by pathological changes and we have recently isolated MVC from the lungs and intestinal tract of a pup in a litter that suffered enteritis and mild respiratory illness. Since more than 50% of neonatal pup illnesses remain undiagnosed, clarification of the role of MVC would be especially helpful to veterinarians and dog breeders.

MVC has proven difficult to isolate from pups and embryonic tissues submitted for necropsy, very likely because of the presence in tissue fluids of neutralizing antibodies to the virus. Dr. Patricia Lucia has cloned a fragment of the genome of MVC into a bacterial plasmid and developed a non-radioactive RNA probe that can detect MVC genomes in infected cell cultures as well as samples from infected pups. Unfortunately, the sensitivity of the current method is lower than that of our virus isolation methods, and more sensitive techniques are now under study. Our immediate goal is to determine the significance of MVC as a cause of reproductive failures and

illness or death in young pups. The disease may be suspected in cases of stillbirths or anasarca, a fetal anomaly.

MVC may also be implicated in cases where young pups from dams immunized against CPV-2 suffer illness with signs of vomiting, diarrhea, and respiratory distress or die suddenly during the first to third weeks of life. Serum samples also are useful, since mothers of ill pups generally have high antibody titers against MVC.

Other Activities

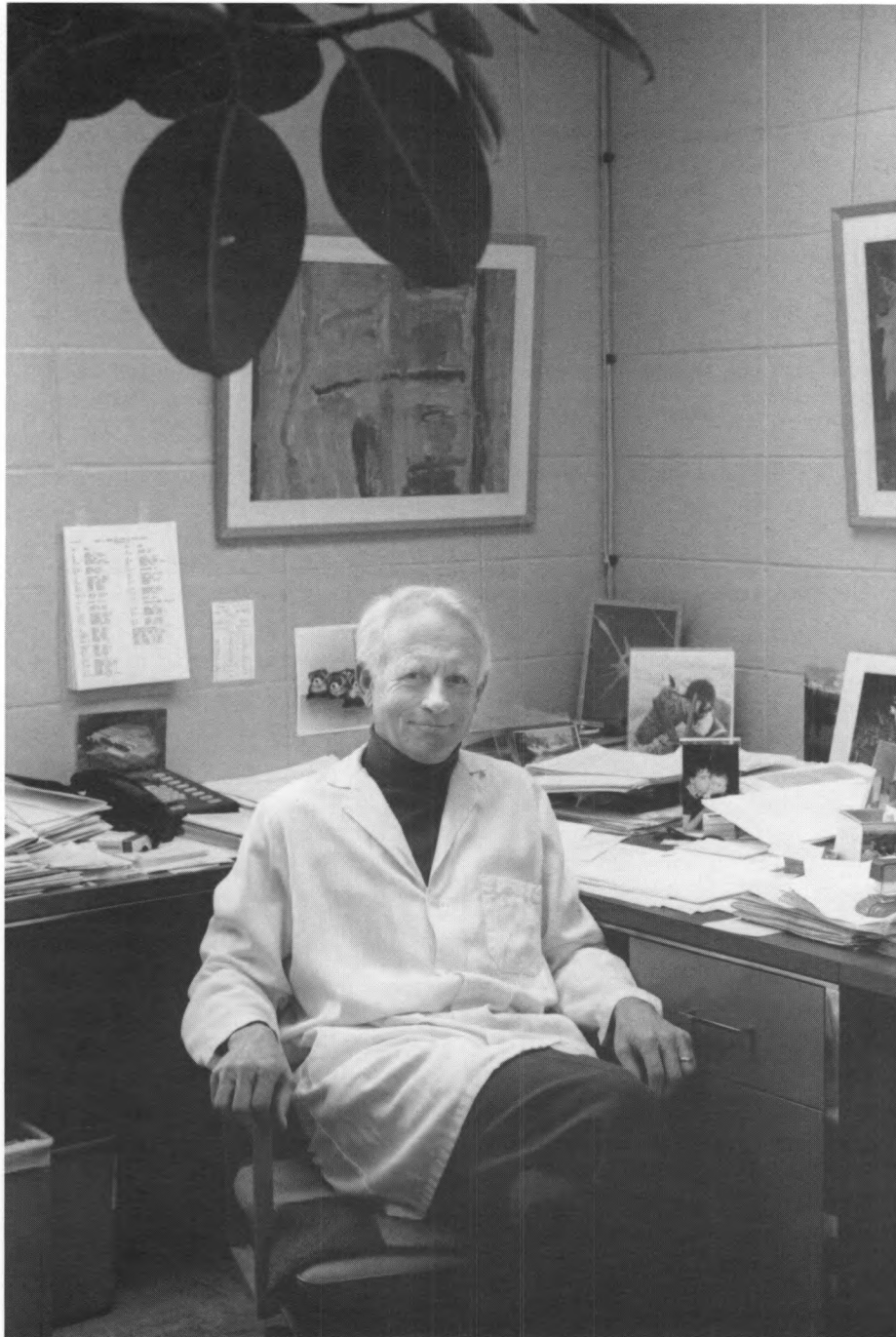
I served as co-chairman of a meeting in Rome, Italy sponsored by FAO, "FAO Expert Consultation on Quality Control of Veterinary Vaccines in Developing Countries". The meeting was attended by more than 50 international experts on the development of livestock vaccines for economically important diseases. In April I served as a member of an advisory council to the dean of the graduate division of the School of Veterinary Medicine of the Universidad Nacional Autónoma de México in Mexico City. We were fortunate to have Amy Kloster join our laboratory, where she now provides cheerful technical assistance.

Minute virus of canines (MVC) may cause fetal resorptions or abortions in pregnant dams and a fatal illness in very young pups. Our immediate goal is to determine the significance of MVC as a cause of reproductive failures and illness or death in young pups.

Leland E. Carmichael

Hadley C. Stephenson Laboratory for the Study of Canine Diseases

DEDE HATCH



Max Appel

Lyme Disease in Dogs

Last year we reported progress in our understanding of the way dogs respond to natural infection with Lyme disease. In studies with *Ixodes dammini* ticks from Westchester County, New York, an endemic area for Lyme disease, we found the greatest susceptibility in young dogs exposed to adult ticks infected with *Borrelia burgdorferi*, the spirochete that causes Lyme disease. For dogs of any age, in fact, the risk of developing Lyme disease is greatest approximately two to five months after exposure to infected adult ticks, which are active in the late fall and early spring months. We also reported that inoculation of cultured *B. burgdorferi* failed to reproduce the disease in dogs, while infected ticks consistently induced infection.

An important goal of our studies is to develop a vaccine that can be proven to be safe and effective in protecting dogs against Lyme disease. But in order to best evaluate the safety and efficacy of potential vaccines, we need to have a controlled means of exposing vaccinated dogs to infection. In our opinion, protection should be tested by the natural route of infection via infected ticks.

Our research team, which includes Sandra Allan from the Department of Entomology and Yung-Fu Chang, Tsai-Ling Lauderdale, Richard Jacobson, Sang Shin, and Brian Summers from the College of Veterinary Medicine, began this year with a study comparing the infectivity of ticks we had raised and infected in the laboratory to that of infected ticks collected in Westchester County. To infect the laboratory ticks, we had allowed the larvae to feed on mice and gerbils infected with a *B. burgdorferi* strain obtained from the Communicable Disease Center in Fort Collins, Colorado. After maturing into the next stage, 80% of the ticks proved to be infected. However, we found



Susan Pearce-Kelling

those infected nymphs unable to infect dogs in turn. That experience showed us the importance of small biological variances on the spirochete-tick-host interaction.

Another study that yielded negative results was an attempt to simulate tick infection using a mixture of *B. burgdorferi* isolated from ticks and tick saliva. It is well known that tick saliva has an immunosuppressive influence on the host immune response and the local microenvironment. Nevertheless, dogs inoculated with the mixture had immune responses similar to those of the dogs that were inoculated only with *B. burgdorferi*, and did not develop clinical signs.

We plan to continue to expose dogs to ticks raised and infected in the laboratory under controlled conditions. For the present time, however, we also continue to collect infected ticks in Westchester County, since they infect dogs with a high rate of success. We have initiated a study to test the immunizing capabilities of selected proteins by vaccinating dogs and later

challenging them by infected tick exposure. We are evaluating vaccine efficacy by using skin samples from the area of the tick bite as a method for isolating spirochetes from dogs. We have found this simple method, which is used in mice and humans, to be reliable for detecting infection in dogs as well.

A highlight of 1992 was the Fifth International Congress on Lyme Borreliosis in Arlington, Virginia, where we reported our results on experimental Lyme disease in dogs. We had several favorable responses to the report following the conference and a comprehensive publication is in press.

Canine Distemper in Large Cats

A major outbreak of canine distemper virus (CDV) in large cats occurred in a zoological park in California this fall. Seventeen animals—including lions, tigers, leopards, and jaguars—succumbed to the disease with respiratory, enteric, and central nervous signs. We believe this to be the first time that CDV has been incriminated in a disease outbreak of proportions great enough to suggest cat-to-cat transmission among large cats. Previously, only three individual cases had been reported.

In collaboration with pathologist Brian Summers, we confirmed the cause of the outbreak by virus isolation, serology, histopathology, and immunocytochemistry. CDV was isolated from seven animals. Monoclonal antibody studies showed all isolates to be classical canine distemper virus. Serum neutralization tests demonstrated the presence of CDV antibody in most of the animals. Histopathology and immunocytochemistry results were also typical of CDV infection.

We can only speculate about the reasons for this outbreak. An unusually large number of CDV-infected raccoons were seen in the park during the year and would seem a likely source of infection. But contact between CDV-infected carnivores and large cats has often been known to take place in zoos in the past without provoking overt disease in large cats. Furthermore, we demonstrated several years ago that domestic cats, although susceptible to CDV infection, did not show clinical signs of disease. In the domestic cats, CDV that was virulent for dogs behaved much like an avirulent distemper vaccine. Why the change now?

One possibility is the emergence of a biological variant of the CDV strain, although our monoclonal antibody studies did not confirm that. Another, perhaps more likely explanation might be that the large cats were also infected with an immunosuppressive agent such as the feline immunodeficiency virus (FIV), which is a lentivirus (or retrovirus) similar to the one that causes AIDS in humans. Although only one of the large cats proved to be seropositive for FIV, it may be that others infected with FIV or another retrovirus had not yet developed antibodies. Other retroviruses are known

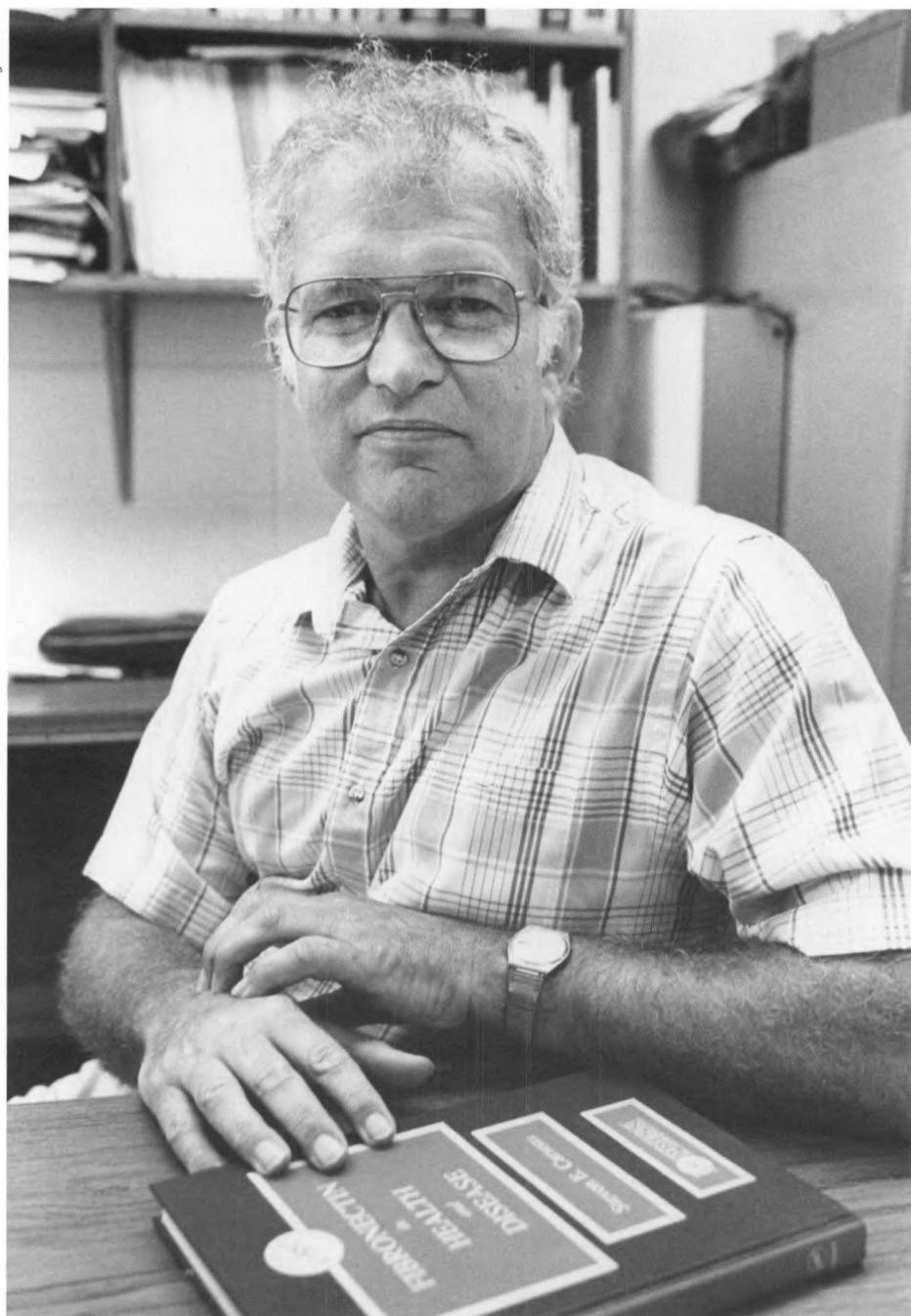
to affect large cats and domestic cats differently. We are collaborating with Dr. Margaret Barr in the Department of Veterinary Microbiology in attempts to demonstrate whether a retrovirus is present in tissues of the affected large cats.

An important goal of our studies is to develop a vaccine that can be proven to be safe and effective in protecting dogs against Lyme disease.

Max J. G. Appel

John M. Olin Laboratory for the Study of Canine Bone and Joint Diseases

JON REIS



George Lust

Hip Dysplasia

The year 1993 will mark the 25th anniversary of the canine hip dysplasia research program at the Institute. It was even longer ago, in 1966, that Sten-Erik Olsson and his colleagues in Sweden first proposed a role for joint laxity in canine hip dysplasia. Although extensive research in several laboratories still has not determined whether laxity is a factor in the development of the disease, it remains an intriguing concept. In collaboration with Gail Smith of the University of Pennsylvania's School of Veterinary Medicine, Alma Williams and I have evaluated a new X-ray procedure for measuring hip joint laxity that Dr. Smith proposed with his colleagues, Drs. Biery and Gregor. We wanted to determine whether this method, which uses a new position of the hip joints during radiologic examination, could predict the later development of hip dysplasia and osteoarthritis in young Labrador Retrievers.

Our study tracked 42 pups from the age of four months to the age of two years. Measurements taken at four months of age showed that pups from dysplastic parents already showed greater hip joint laxity than pups from normal parents. Follow-up studies indicated that relatively tight joints (a laxity of less than 3.0mm) at four months predicted normal hip joints in about 90% of the cases, while a laxity greater than that correctly predicted hip dysplasia in 57% of the dogs. Using a method of statistical analysis represented in the adjacent graphs, we found that the probability of a dog having normal hip joints decreases with increasing hip joint laxity. We also found that a normal or dysplastic outcome can be better predicted at eight months of age than at four months.

In March of 1992 I attended the annual conference of the Veterinary

Orthopedic Society in Keystone, Colorado and presented the results of our studies showing that injections of Adequan® can be helpful in preventing the development of hip dysplasia in growing dogs. In September I took part in a mini-course entitled "Comparative Aspects of Hip and Knee Lesions in Dog and Man" that was convened in Uppsala, Sweden to commemorate the retirement of Sten-Erik Olsson and honor him for the many important contributions he has made to the field of veterinary radiology and orthopedics. I have known Professor Olsson as a colleague and friend since 1969 and have valued his insights during many useful and interesting discussions of hip dysplasia in dogs.

In our continuing collaboration with the Ciba-Geigy Corporation, we are studying the role of cartilage enzymes that act at neutral or physiological pH during the development of osteoarthritis. Labrador Retrievers can develop spontaneous osteoarthritis in their hips in addition to hip dysplasia. Our role in the cooperative effort is to test new drugs in such dogs to determine whether they have promise for treating or even eliminating hip dysplasia.

One class of enzymes that work at

We want to determine whether a new method of measuring hip joint laxity in young dogs can predict the later development of hip dysplasia and osteoarthritis.

neutral pH, stromelysins, destroy the structural protein of the joint cartilage as osteoarthritis develops. We are now examining stromelysins to determine their role

in canine osteoarthritis. We have demonstrated in tissue culture that the action of the stromelysins is triggered

by cytokines, a protein released by stimulated white blood cells (leukocytes). The leukocytes release cytokines in response to a variety of abnormal stimuli, including the osteoarthritic state, inflammation, and infection. The observation of the role of cytokines in the destruction of joint cartilage will help us to test the culture medium and the cartilage for stromelysin or other similar enzyme activity. If the enzyme activity is present, we can then test in culture a new class of drugs being developed that inhibit these enzymes. If those tests are successful, we will conduct trials in dogs with osteoarthritis. Because these drugs are inhibitors of specific enzymes, it is expected that they may be more effective in preventing or slowing the progression of osteoarthritis than the medications in current use, which merely treat symptoms.

Rory Todhunter travelled to the University of Lund, Sweden in May to deliver samples from dogs with hip dysplasia to Dick Heinegård's laboratory, where several new antibodies are available that recognize breakdown products of the cartilage matrix. A PhD student in Dr. Heinegård's laboratory is using the samples in studies intended to follow the osteoarthritic process from its early stage to the time that actual disease develops in order to compare the pathogenesis of the human and canine diseases.

Dr. Todhunter and Elizabeth Grisanzio also have continued studies aimed at discovering a genetic marker for hip dysplasia. We are searching white blood cells for a fragment of the DNA molecule that will allow us to differentiate between normal pups and those dogs that are at high risk of developing dysplastic hip joints.

George Lust

Figures 1 and 2 show a statistical logistic regression model relating measurements of hip joint laxity in puppies to the probability of a dog having normal hip joints as an adult. Laxity is represented in terms of a ratio, called the distraction index, that represents the distance that the femoral head is displaced from the center of the acetabulum divided by the radius of the femoral head.

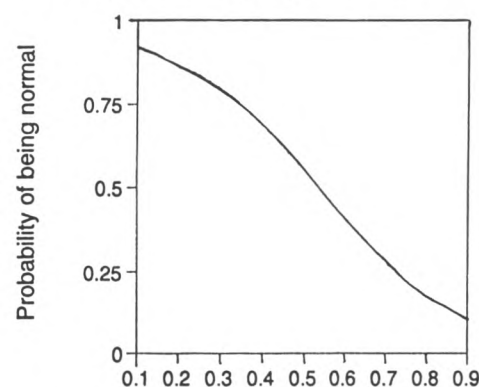


Figure 1. Laxity measured at four months of age: the probability of a dog having normal hip joints later in life decreases with increasing hip joint laxity.

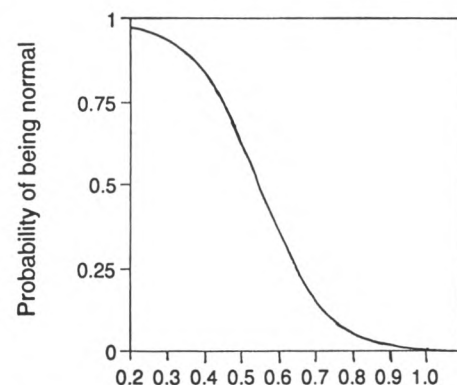


Figure 2. Laxity measured at eight months indicates that a normal or dysplastic outcome later in life can be better predicted after the age of four months. Both graphs suggest that an index greater than 0.4 is not a sufficiently reliable basis for a clinical diagnosis of canine hip dysplasia. This appears to be especially true for distraction indexes between 0.4 and 0.7. An index greater than 0.7 at eight months is much more likely to be associated with dysplastic joints. On the other hand, a distraction index below 0.3 predicts a high probability for normal joint development.



Nancy Burton-Wurster

Osteoarthritis

In the 12 years since I began work with George Lust studying the osteoarthritis and cartilage abnormalities that accompany canine hip dysplasia, we have approached the study of cartilage degeneration in osteoarthritis from two perspectives. First, we and others have striven to understand the fundamental

composition and biology of normal cartilage and the aberrations that occur in diseased tissue. In particular, our laboratory has focused on the important extracellular adhesion molecule, fibronectin. Our second approach attempts to mimic some of the characteristics of degenerating cartilage in osteoarthritic joints using

tissue and organ culture methods. To do this, we have manipulated several biochemical and mechanical parameters to test for changes in hydration, chemical composition, biosynthetic patterns, and structural integrity that might signal early stages of degeneration.

Fibronectin is a key molecule in several important biological processes including embryonic development and wound healing. Although we don't yet know the specific function of fibronectin within cartilage, we know that it accumulates markedly in osteoarthritic tissue. We have identified some unique structural features of cartilage fibronectin that we believe influence its role in that tissue. At the Society for Cell Biology meeting held last November in Denver, I presented our finding that some cartilage fibronectin contains a modification not found in fibronectins of blood or, as far as we know, in other tissues. This modification consists of the addition of a glycosaminoglycan (GAG) side chain. This makes the protein more complex and likely to function as the matrix or tissue "glue" that surrounds the individual cartilage cells.

The presence of a GAG chain is not the only unusual feature of cartilage fibronectin. There are small variations in the sequences of amino acids that compose the different types of fibronectin. Fibronectin containing the sequence termed "ED-B" is present in various tissues in the early embryo, but it is not commonly found in adult tissue. However, Dai Wei Zhang, an orthopedic surgeon studying in our laboratory, has demonstrated that there is a high level of ED-B messenger RNA in adult cartilage fibronectin. (The messenger RNA is the means by which the cell translates the genetic code and forms a protein.) Dr. Zhang now plans to make antibodies that will bind to the ED-B sequence in fibronectin. Tagged with a color-producing enzyme or a

We have identified some unique structural features of cartilage fibronectin that may influence its role in that tissue.

fluorescent marker, those bound antibodies will permit him to identify and determine the quantity of ED-B positive fibronectin

in normal and osteoarthritic tissue, and also to detect any release of cartilage fibronectin into the blood as a result of disease-related degeneration. In February of 1993 Dr. Zhang and I will attend the Orthopaedic Research Society meetings in San Francisco, where his abstract describing this work will be presented.

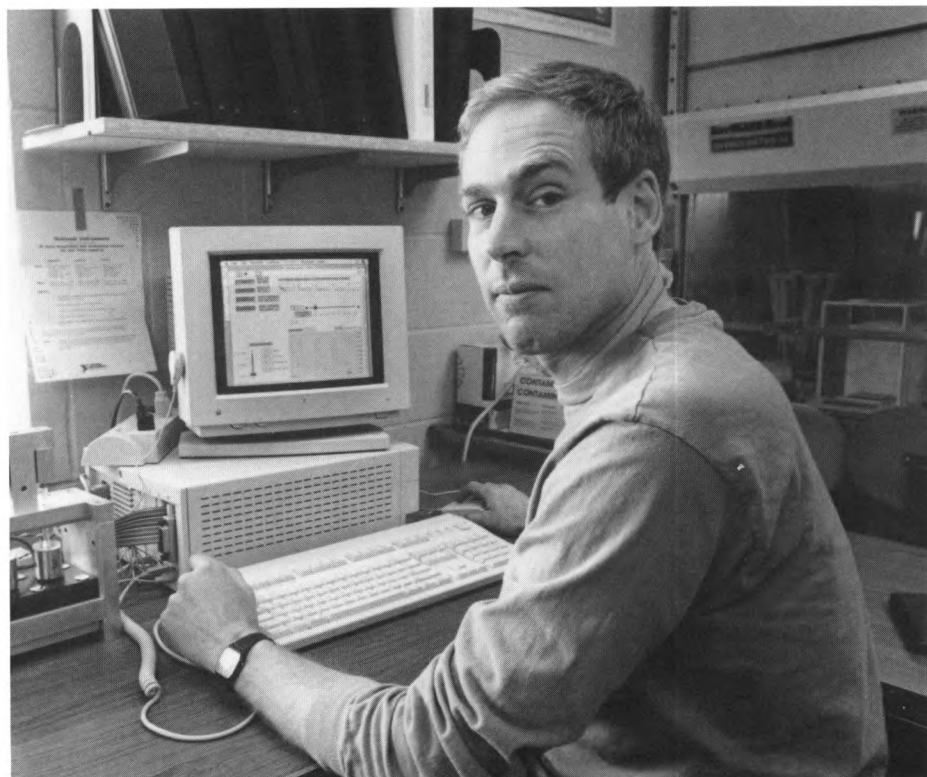
Tony Farquhar, a mechanical engineer whose PhD thesis involved the development of a computer model to describe the micro-mechanical response of articular cartilage to a mechanical load, joined our laboratory last year as a post-doctoral associate with the goal of testing the hypothesis, derived in part from his model, that certain loading protocols will induce damage in the extracellular matrix. Such damage would be expected to induce degenerative changes. To do this, he has developed a novel testing device capable of supplying dynamic loads of up to five times body weight to cartilage. This work is done using isolated cartilage discs and is aimed at replicating in the laboratory what actually happens in the dog with a normal hip joint as compared to the dog with a predisposition to hip dysplasia. Dr. Farquhar and Margaret Vernier-Singer have completed the first tests of this new apparatus, and while much remains to be done, initial results have revealed some intriguing differences between the response of the central core and the peripheral ring of the cartilage discs.

In parallel biochemical experiments, we have continued to study a regulatory protein, TGF-B. We already knew that TGF-B would markedly increase the production of fibronectin in isolated cartilage grown in organ culture. Our first attempts to mimic in culture the retention and accumulation of fibronectin that occurs within the matrix of functioning osteoarthritic cartilage did not achieve the desired effect, as most of the increased fibronectin was lost to the culture medium. We solved that problem by adding fucoidan, a compound found in marine algae that appears to encourage the retention of extracellular matrix proteins including fibronectin. We have found that, by using fucoidan and TGF-B in combination, we can achieve a significant accumulation of fibronectin in the matrix of cultured normal cartilage. We must now test our hypothesis that this accumulation will

contribute to other degenerative changes in the cartilage.

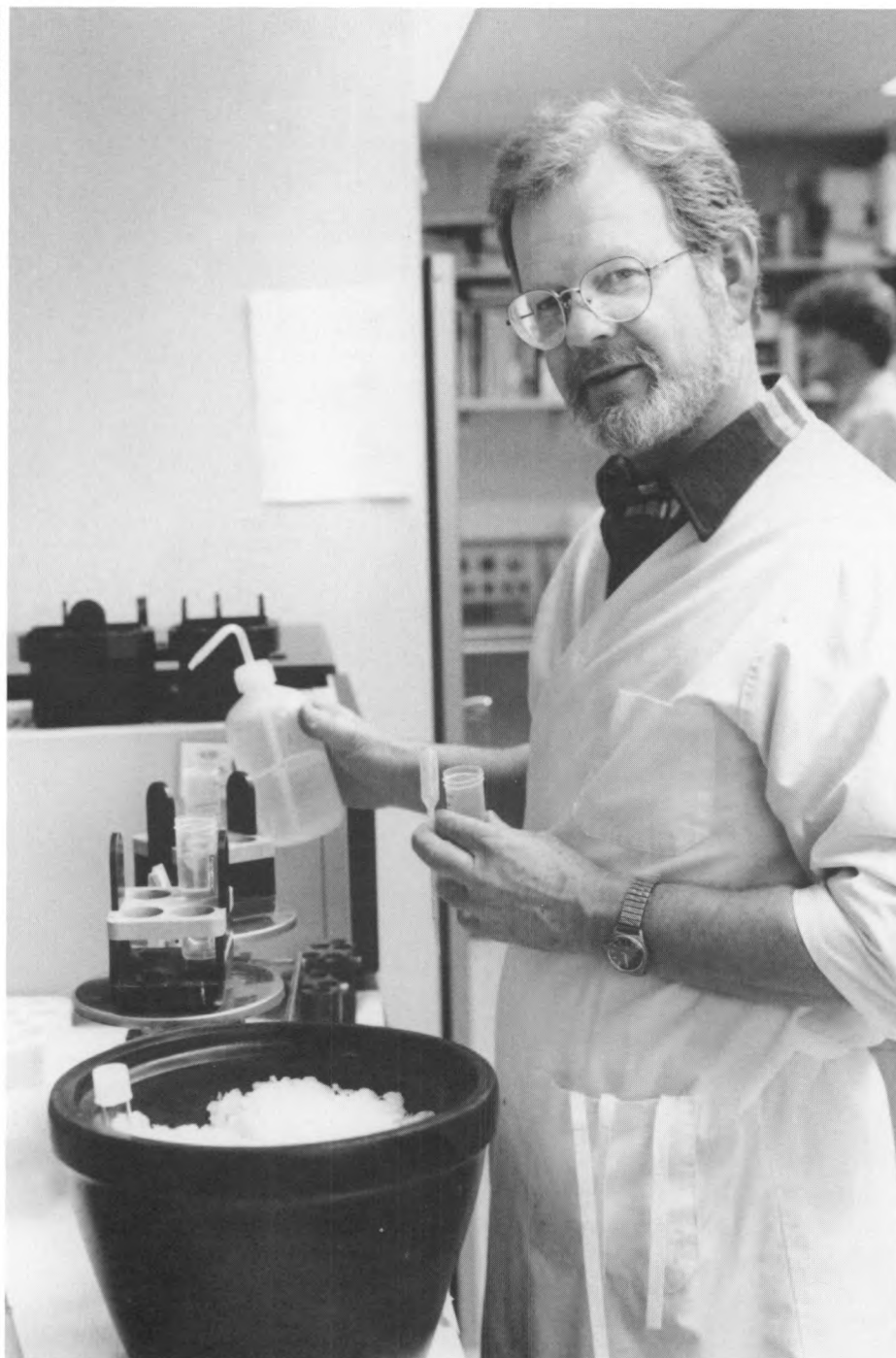
My husband and I enjoyed a trip last spring to England and Germany. While in Germany, we visited with former post-doctoral fellow Juergen Steinmeyer, who has continued the work he began at the Institute in his own laboratory at the University of Bonn. In England, I visited the London laboratory of Robert Brown, whose visit to Ithaca a few years ago spurred the work that eventually led us to look for a glycosaminoglycan structure on cartilage fibronectin. I also visited the Manchester laboratory of Shirley Ayad, who has made significant contributions to the study of type VI and type IX collagens in cartilage. Dr. Brown and Dr. Ayad were cordial hosts, and we had a wonderful time.

Nancy Burton-Wurster



Anthony Farquhar

DEDE HATCH



Robin Bell

A Beneficial Role for IgE

Most of us know someone who suffers from hay fever, food allergy, asthma, or some other manifestation of allergic disease. Similar diseases can be a major problem in domestic animals, including dogs, where skin allergies, frequently to fleas, are prominent. Most of these reactions are caused by an antibody molecule of a specific type, IgE, which binds preferentially to mast cells, a specialized cell common in skin, the lung, and the intestine. When IgE on the surface of mast cells in the skin or lungs encounters its target antigen, the allergen, the mast cells degranulate and release several biologically active substances that cause the red, itchy, raised inflammatory lesions characteristic of skin allergies. This biological sequence has been recognized for many years, although mostly in the negative effects of unwanted allergic reactions. The puzzle has been in recognizing the beneficial effects of IgE and mast cell degranulation. No one thinks hay fever is a "good thing"; if, as it is believed, immune mechanisms evolve to bring benefit and not misery, what good does IgE do?

Several years ago, investigators in this laboratory were able to demonstrate an important function for IgE in resisting infection by parasites. In those experiments, we collected serum from rats that had been infected with *Trichinella spiralis*, the nematode parasite that causes trichinosis in man and several animal species. We then isolated pure IgE from the serum. When the IgE, which contained specific antibody to *T. spiralis*, was transferred to rats that had never encountered the parasite before, we were able to show specific rejection of the worm. Those experiments were important because they showed an unequivocal function for IgE in a response-parasite

rejection—that is beneficial for the host. In describing this reaction I have greatly simplified the system we used. An experimental prerequisite for obtaining functional IgE responses leading to parasite rejection was that we first transfer specifically activated lymphocytes (white blood cells), then the IgE. Without the prior transfer of the lymphocytes, the IgE was not able to protect the animals, even though it was present in adequate amounts. The biological synergism represented by this two-step is therefore absolute and it tells us that this particular immune response is complex and requires the cooperation of different elements of the immune system.

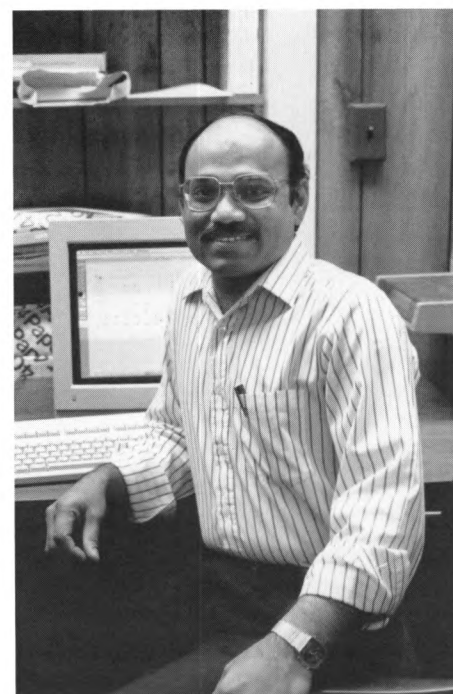
Kalyanasundaram Ramaswamy, a veterinarian who joined our laboratory this year after completing a PhD in respiratory tract immunology in Calgary, Alberta, Canada, is attempting to determine how the IgE functions in this system. To do that he has been using an IgE myeloma protein, IR162, labeled with the radioisotope ^{125}I . We inject the ^{125}I -IgE and measure its concentration in various tissues and organs. This procedure has enabled us to determine where IgE goes as well as the rate of its accumulation in various

sites in the body. The data have shown that there is an important IgE concentrating mechanism in the intestine. Although this process was statistically meaningful in normal rats, it has been

difficult to quantify the events since only about 2% of the total injected ^{125}I -IgE could be accounted for. Nevertheless, this was proportionately more than the amount of IgG transported to the intestine after intravenous injection of ^{125}I -labelled IgG. Such data indicate that we are dealing with an IgE-specific transport mechanism.

To determine whether this transport mechanism might play a role in the rejection of parasites, we measured IgE transport during the period of infection. We found that transport increased during the first two weeks of infection but declined to resting levels by the fourth week of infection. This was very interesting, because it suggested that the IgE transport mechanism was inducible; in other words, it could increase its capacity in response to an infection. To examine this possibility further, we collected the same immune lymphocytes previously used to demonstrate protection with IgE, injected the cells, and then measured ^{125}I -IgE transport. The transfer of cells produced a 100% increase in the amount of IgE transported into the intestine. Although we have not yet proved it, it is plausible to infer from these experiments that the role of the cells in our “cells and IgE” protection system is to increase IgE transport in the gut, thereby regulating rejection of the parasite. We believe that this work has demonstrated an inducible IgE-specific transport mechanism that is present in the intestine and important in host defenses. This discovery is important for understanding not only the mechanisms of immunity to *T. spiralis*, but also general protective and allergic responses, whether they occur in the bowel or in the respiratory tract, or in dogs or in humans, because basic allergic mechanisms appear to be shared by mammals in general.

We have demonstrated a mechanism that is important for understanding not only immunity to *Trichinella spiralis*, but also general protective and allergic responses in all mammals, including dogs and humans.



Kalyanasundaram Ramaswamy

Other Activities

In July I was invited to lecture in the Biology of Parasitism course at the Woods Hole Biological Research Laboratories in Massachusetts. In August I had the opportunity to visit Eastern Europe and deliver lectures at the Seventh International Congress of the Society for Mucosal Immunology in Prague, Czech Republic and the Eighth International Congress of Immunology in Budapest, Hungary. Both meetings attracted a great number of scientists from America and Western Europe.

Robin G. Bell

Mucosal Immunity Laboratory

DEDE HATCH



Judith Appleton

Trichinella spiralis

I spent the 1991-92 academic year on sabbatical in Oxford working with Don Mason in the Cellular Immunology Unit. There I began a new series of investigations into the regulation of antibody production in parasitized animals. For these studies we have used rats as a model for understanding mechanisms of intestinal mucosal immunity that are common to many other animal species.

B lymphocytes are the immune cells in mammals that produce antibodies against infection. In order to perform this function, however, they require the assistance of "helper" T lymphocytes.

For example, although the B lymphocytes produce the antibodies that protect young rats against infection with *Trichinella spiralis*, T lymphocytes influence the kinds of antibodies the B cells will produce. They do this by releasing cytokines, the factors that stimulate B cells to produce antibodies. Cytokines have been under intense scrutiny in many laboratories in recent years, but little is understood about how the T lymphocyte comes to release them. With the help of Lucy Gagliardo and Jeb Oblak, I am continuing the research I began in Oxford to characterize the T lymphocytes involved and the influence that adrenal hormones have on the T cells.

There is other progress to report in our biochemical and genetic investigations of the *T.*

spiralis parasite molecules that are targeted by protective antibodies. Prema Arasu has isolated two recombinant parasite genes and purified their proteins. In collaboration with Florencio Ubeira of the University of Santiago de Compostela in Spain, we have determined that both proteins are present in granules in the secretory organ of the parasite. However, the two proteins have distinct and complementary distributions in those granules. Dr. Arasu has found that immunization with one of the two proteins induces the production of protective antibodies. These results hold significance for the development of vaccines against other infectious diseases that begin in the intestine.

In other experiments, Lauri Ellis has determined that the monoclonal antibodies we have studied protect by binding to sugars attached to the parasite molecules. We are collaborating with Anne Dell in the Imperial College in London to determine the structure and composition of those sugars. We hope that this information will help us design further experiments to elucidate the mechanism of protective immunity against *T. spiralis*.

Equine Influenza Virus

Laura Hanson has continued her work on antigenic variation in the equine influenza virus hemagglutinin. She has found that the viruses isolated in 1985 more closely resemble the viruses isolated in 1963 than those isolated in the 1970s. This phenomenon is due to mutations sustained by the virus. Those mutations caused the molecule to react in serologic assays more like the 1963 viruses, even though other mutations prove that the recent viruses have evolved

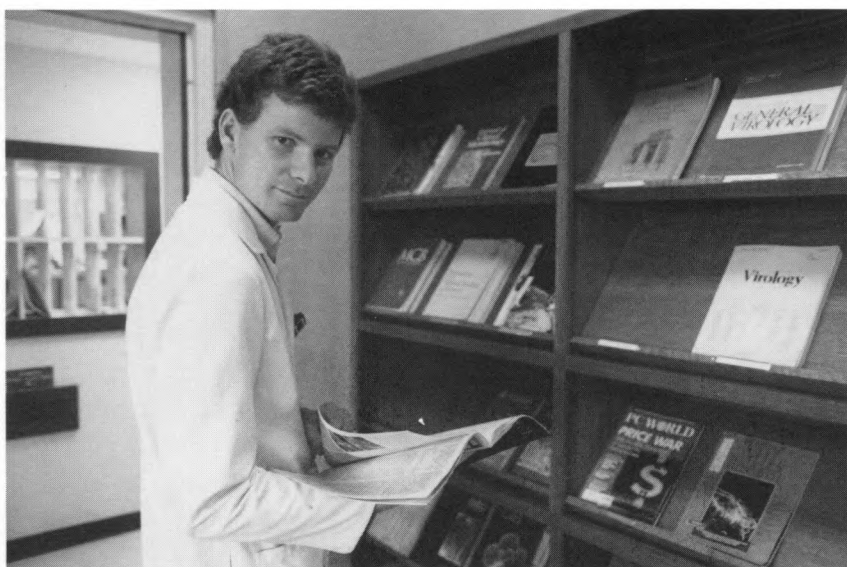
from the newer ones circulating in the 1970s. This "antigenic reversion" is a novel finding. In contrast to human influenza vaccines, which are quite effective, equine influenza vaccines fail to confer lasting immunity. Continued surveillance of influenza for mutation is critical to the improvement of vaccines.

***We want to
determine how T
lymphocytes come to
release cytokines, the
factors that stimulate
B cells to produce
antibodies in
response to infection
with Trichinella
spiralis.***

Judith A. Appleton

Bostwick Laboratory of Molecular Biology

DEDE HATCH



Colin Parrish

Cornell hosted this year's annual conference of the American Society for Virology. The members of this laboratory were involved in the organization and operation of the conference, which was a great success with over 1,800 virologists from around the world attending.

Canine Parvovirus

In the past year we have extended our efforts in new and intensive studies of the genetics and structures of canine and feline parvoviruses. We have been able to acquire new equipment and facilities that have allowed us to make significant strides in both our molecular studies, which are defining the functions of the dog and cat viruses, and in our applied studies, which are aimed towards preparing a new generation of canine parvovirus (CPV) vaccine.

Our efforts to examine the fundamental nature of CPV have continued to shed light on the way in which the virus interacts with the host cells it infects. In particular, we are interested in the properties of the virus that allowed it to emerge as a canine pathogen. To that end, we are

comparing it to the closely related parvovirus of cats, feline panleukopenia virus (FPV). We showed that changes in only two or three amino acids in the protein that makes up the coat of the virus particle distinguish CPV from the feline virus and allow it to replicate in dog cells. Since we know where the atoms of the proteins fit into the structure of the particle, we can tell that those changes are mostly on its surface.

Recently we have completed a project with our colleagues at Purdue University to determine the structure of the FPV capsid. That work shows that the changes we have defined as affecting the host ranges of the viruses also cause the protein chains to fold differently. The locations of the changes within the structures of the viruses have given us new insights into their possible functions. Most likely, they alter the ability of the virus particles to come apart and release their genetic material during the process of infecting the cell. This is the critical stage in the infectious process when the virus coat protein—which is able to survive for months in the environment—must fall apart in a very specific way within a few minutes of entering a cell. In our current studies

we are trying to determine whether the various changes we have defined as affecting the virus's ability to infect dog

cells in culture operate through the same mechanism, and whether the process involves a disassembly step.

We have also initiated studies of the mechanisms by which the virus binds to the outside of the cell so that it can infect it. In those studies, Dina Barbis has been able to show that the virus coat protein binds to certain sugar molecules on host cells and on certain red blood cells in a very specific way. Dr. Barbis is also studying the virus's interactions with other cell-surface proteins in an effort to characterize the cellular protein that allows infection to take place.

In a separate study with Leland Carmichael, we have derived an attenuated strain of CPV-2b, the new antigenic variant of CPV commonly found to be infecting dogs in the United States. Allen Gruenberg has been able to clone the genome of the attenuated virus into a bacterial plasmid. In that form the virus's genetic material is not subject to the further selection that occurs during extended passage of a virus in tissue culture. Dr. Gruenberg is determining the DNA sequence of the attenuated CPV-2b genome in order to explain why the virus does not cause disease in dogs. This is the first time that a vaccine strain of any parvovirus has been so well characterized. We believe that this work will form the basis of a new generation of safe and stable CPV vaccines that will vaccinate dogs efficiently against the current strains of CPV.

We have made significant strides both in our molecular studies, which are defining the functions of the dog and cat parvoviruses, and in our applied studies, which are aimed towards preparing a new generation of canine parvovirus vaccine.

Colin R. Parrish

Equine Genetics Center

This was an important transition year for the Equine Genetics Center. Several research projects have added molecular biology approaches to our armament of cell biology and immunology techniques. The extra efforts have opened new vistas for further research in equine immunology, genetics, and reproduction.

Gene Jockeys and Horse Genetics

The two principal projects that use molecular techniques in our laboratory are complementary investigations of the relationship between pregnancy and histocompatibility in the mare. With support from the Zweig Memorial Fund, Juli Maher is studying the molecular mechanisms regulating the expression of histocompatibility

Understanding how the equine trophoblast switches on and off the expression of paternal MHC antigens could shed light on the problem of abortion in mares and also have important implications for human organ transplantation and in the development of new immunological treatments for cancer.

therefore forms the barrier between mother and fetus. The portion of equine trophoblast that invades deep into the uterus expresses high levels of

antigens in the placenta. In the horse we have observed a very unusual pattern of expression of the Major Histocompatibility Complex (MHC) class I antigens in the trophoblast, the tissue that makes up the outer layer of the placenta and



Douglas Antczak

paternal MHC antigens, but only for a short period between days 30 and 45 of gestation. As a result of this high level of MHC antigen expression, the mare mounts a strong immune response against these fetal antigens. Understanding how the trophoblast switches the MHC genes on and off could shed light on the problem of abortion in mares and also have important implications for human organ transplantation and in the development of new immunological treatments for cancer. Dr. Maher has been awarded a three-year postdoctoral fellowship from the National Institutes of Health (NIH) for her studies.

In the second project, Gabriele Grunig is pioneering the application of molecular probes for cytokines to studies of immune cell function in the uterus of the mare. Cytokines are small molecules that direct the differentiation and function of lymphocytes, or white blood cells. They are secreted by cells of the immune system, often in low concentration and only in restricted areas. Dr. Grunig is using a technique known as *in situ* hybridization to detect molecular

messages within the cells in the pregnant uterus that determine the production of equine cytokines. This work has potential therapeutic use beyond its current focus on how the mare responds to the antigenic challenge of pregnancy; the availability of cloned genes for various equine cytokines would lead to the production of pure preparations of those molecules that might be used, for instance, in treating cases of immunodeficiency caused by poor nutrition, infection, or other environmental factors. Dr. Grunig's research is supported by a grant to our laboratory from the National Institute of Child Health and Human Development of the NIH.

In other studies, we are continuing our efforts to maintain and differentiate individual equine trophoblast cells in culture and searching for ways to isolate lymphocytes from the equine uterus for studies of their composition and function. Both of these goals have proved difficult to achieve, but they are critical to the advancement of the projects described above.

Havemeyer Foundation Symposia

We participated this year in the organization of two small conferences convened by the Dorothy Russell Havemeyer Foundation. The first was an international meeting on Equine Neonatal Medicine held in January in Naples, Florida. The meeting was attended by an enthusiastic group of veterinary clinicians, basic scientists, and physicians. The second meeting, the Third International Symposium on Equine Embryo Transfer, was held in Buenos Aires, Argentina in November.

Julio Oriol, an Argentine veterinarian who completed the MS degree in this laboratory in 1988, was a principal organizer of this highly successful meeting.

Embryo transfer has been less successful in horses than in other species, in large measure because of the difficulty of obtaining large numbers of embryos from a mare during a single estrus cycle. Procedures and techniques that work well to achieve so-called "super ovulation" in most other species, including humans, have not worked when applied to the horse; it is extremely unusual to collect more than a single fertilized egg from any donor mare during any estrus cycle. And the seasonal breeding behavior of horses is such that there is only a six-month period during which mares can be easily mated and embryos collected. It is unusual to collect more than three embryos per year per donor mare. With a transfer success rate between 50% and 75%, this means that an owner can expect only one or two foals from very fertile donor mares each year.

Given these and other problems, the need for further research on equine



Jane Miller and James Hardy

embryo transfer should be apparent. The symposium in Buenos Aires did not bring solutions to all the outstanding problems of equine reproduction, but the science presented was excellent, the location superb, and the hospitality of our Argentine hosts unparalleled. The participants had the opportunity to visit the School of Veterinary Medicine at the University of Buenos Aires and to attend matches of the Argentine Open Polo Tournament, an unofficial "world championship". We also were able to visit Thoroughbred breeding farms and race tracks, and *estancias* where *gauchos* still rule on the beautiful Argentine *criollo* horses.

Summer Fellows and Other News

The Equine Genetics Center-Havemeyer Foundation Summer Fellows program for veterinary students continues to attract large numbers of highly qualified applicants. Leslie Triplett of Oregon State University and Julie Stanek of Texas A&M were chosen

from a pool of 23 applicants from 15 veterinary schools to join us in a productive summer of research.

Graduation at Cornell was a very special time this year, with three graduate students from our laboratory returning to campus to take their degrees. Anne Crump and Chonghui Zhang are now postdoctoral fellows at the Dana Farber Cancer Institute of Harvard Medical School. Bill Donaldson works in Regulatory Affairs for the Animal Health and Agriculture Research and Development Division of Merck & Co.

In April we organized a one-day scientific meeting in honor of Dr. S. J. Roberts, an emeritus professor of veterinary obstetrics and former coach of the polo team at Cornell. The meeting was held in conjunction with a celebration of the 20th anniversary of Dr. Roberts's retirement from Cornell and was organized by the Cornell Polo Club.

D. F. Antczak

Laboratory of Cellular Growth and Differentiation

DEDE HATCH



James MacLeod

Having joined the Baker Institute faculty in June, this is my first contribution to the annual report. My scientific interests focus on the process of cellular differentiation and on defining specific molecular functions of differentiated cells. This knowledge is important in clinical medicine, because many diseases interrupt the specialized functional properties of differentiated tissues. I would like to take this opportunity to introduce two new studies that are now in progress.

Bone Growth

The first project involves the study of cellular and molecular regulators of long bone growth and is being conducted in collaboration with Dr. Cornelia Farnum in the Department of Anatomy. Long bones, such as the femur or tibia, lengthen from their ends by interstitial growth. The growth process involves cells called chondrocytes that are organized along epiphyseal growth plates and synthesize a cartilaginous matrix. This matrix is subsequently mineralized and remodeled to form new lamellar bone.

Our research plan is to compare chondrocytes in three strains of mice that grow at markedly different rates due to altered growth hormone expression. The first strain is a dwarf line, containing a genetic mutation that blocks growth hormone production. The second strain is a normal control. The third strain grows to a significantly larger size than normal due to extra and stably inherited copies of the growth hormone gene. On a cellular level, we will measure the total pool of dividing chondrocytes and determine their replication rate using chemical markers of DNA synthesis. We will also compare changes in chondrocyte volume and height, normally an important contributor to growth that occurs after the cessation of cell division. On a molecular level, we will

Our purpose is to identify important regulators of linear bone growth and gain insights into the mechanisms of growth hormone action. The results of this study should be applicable to a further understanding of bone growth in all mammals, and particularly in dogs, where size differences can be remarkable.

called *in situ* hybridization. Basically, radiolabeled DNA specific for the gene under study is incubated with very thin longitudinal sections of growth plate tissue. In areas where the gene is actively expressed, the DNA probe will bind. Energy released by the radioactive decay of the probe is recorded on film and can be directly localized to individual cells. Combined, these cellular and molecular techniques will generate parallel profiles of the events occurring during chondrocytic differentiation. Our purpose is to identify important regulators of linear bone growth and gain insights into the mechanisms of growth hormone action.

Scientifically, the advantage of conducting this study using inbred strains of mice is the ability to minimize other experimental variables that are not being studied. Except for growth hormone, the mice are nearly identical genetically. Environmental variables are easier to standardize due to the small body size of mice. Because the basic mechanisms of growth and development are largely conserved in evolution across species, results collected in this study should be applicable to a further understanding of both normal and abnormal bone growth in all mammals, and particularly in dogs, where there is great variability

measure changes in the expression of several genes that encode important matrix proteins and growth factors. The information will be collected using a technique

in size between breeds such as the Chihuahua and the Mastiff. Size differences can be remarkable even within one breed of dog, as for instance in Toy, Miniature, and Standard Poodles.

Synovial Lining Cells and Synovial Fluid

Cells lining the inner surface of the tissue capsule surrounding movable joints synthesize and secrete many of the specialized components present in synovial fluid. A molecule of particular interest is a glycoprotein that has been reported to provide important lubricating properties for the joint surfaces. To better understand the structure and function of this protein, we hope to isolate and clone its gene. We intend to begin by purifying the protein from synovial fluid and analyzing portions of its amino acid sequence. The sequences we identify will provide clues to the composition of the corresponding segments of the gene. Once we have deduced those small sections of DNA, we can use them to locate the entire gene. The process is full of technical difficulties, beginning with developing a functional lubrication assay that will accurately represent joint biomechanics and enable us to achieve the initial purification of the glycoprotein. To this end, we are hoping to use a human hip prosthesis in an apparatus that measures the friction generated by different test fluids.

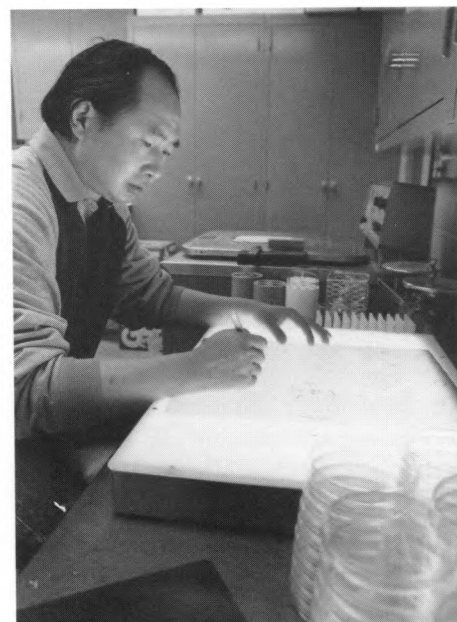
Our laboratory is also planning to proceed with a more general approach called subtractive library hybridization. Rather than targeting a specific gene from the beginning, this method attempts to identify important proteins in a population of cells by comparing their messenger RNA to that expressed in a second cell population and eliminating all the molecules that are common to both preparations. The unique sequences that remain are then

isolated and analyzed using standard molecular biology techniques. This unbiased approach relies on the experimental results to identify important molecules for further study.

Changes in synovial fluid occur in most forms of joint pathology. It is often unclear if these changes are a cause or an effect of the ongoing disease process. By studying synovial lining cells and their protein products, we hope to address this question and better understand synovial fluid's functional role in joints.

In closing, I would like to introduce Da-Nian Gu, a postdoctoral associate who joined my laboratory in September. Dr. Gu received his training at the Genetics Institute of Fudan University in Shanghai, China. Prior to coming to Ithaca, he spent two years as a postdoctoral fellow at the Uniformed Services University of the Health Sciences in Bethesda, Maryland. I would also like to express my sincere gratitude to all of the Institute personnel who have been so helpful and supportive during my first year at Cornell. I am honored to be here.

James N. MacLeod



Da-Nian Gu

DEDE HATCH

Laboratory for the Study of Inherited Canine Reproductive Diseases



DEDE HATCH

Vicki Meyers-Wallen and Olga

We moved our research program to the Baker Institute in January. In addition, we have a new technician, Vicky Palmer, who performs special immunostaining and molecular techniques and procedures essential for karyotyping. We have continued our studies of inherited abnormalities of sexual development in dogs, focusing on persistent Müllerian duct syndrome (PMDS) in the Miniature Schnauzer and XX sex reversal in the American Cocker Spaniel and the German Shorthaired Pointer.

Persistent Müllerian Duct Syndrome

The development of normal male phenotypic sex, that is, the visible expression of male traits, depends on the secretion of two testicular hormones, testosterone and Müllerian-inhibiting substance (MIS). MIS is a glycoprotein that blocks the formation of a female reproductive system in the developing male embryo. It does this by causing the regression of the Müllerian duct system, which is present in all

embryos up to this stage of sexual differentiation. When the Müllerian duct system persists, as it does in females, it develops into the uterus and oviducts.

We have been studying a line of Miniature Schnauzers in which an inherited reproductive defect allows a uterus to develop in otherwise normal males. We want to determine whether the hormone MIS is absent or whether the Müllerian ducts are failing to respond to its presence. We have pinpointed the gestational age at which Müllerian duct regression occurs in normal males and found that, at that age, MIS production is the same in normal and PMDS-affected males. But despite normal production of MIS, we found that the PMDS males still had Müllerian ducts. These findings suggest that the Müllerian ducts persist in male dogs that inherit PMDS because the ducts are unable to pick up the signal being sent by the MIS. This hormone is believed to act through a specific receptor, and it is there that we now think the defect responsible for PMDS may reside. Current studies aim to identify and characterize the MIS receptor.

XX Sex Reversal

In this inherited defect of American Cocker Spaniels and German Shorthaired Pointers, testicular tissue develops in dogs that have the chromosome constitution of females. In addition, the Müllerian ducts fail to regress, and a complete uterus develops. Our studies during the past year suggest that testicular tissue forms much later in XX sex-reversed embryos than in normal male embryos. It is likely, then, that MIS is produced too late to induce regression of the Müllerian ducts, with the result that the ducts remain and develop into a uterus. Further studies will use molecular techniques to confirm these findings and to investigate the basis for testicular development in XX sex-reversed dogs.

Other Activities

I was invited to lecture on inherited abnormalities of the reproductive system in dogs and cats last summer at the International Congress of Animal Reproduction and Artificial Insemination, which was held in the Netherlands, and the Second International Conference on Canine and Feline Reproduction, in Belgium. Following those meetings, I repaired to France, where I visited the Loire Valley and the Normandy coast and took in all I could of Paris. Paris may require further study at a later date!

*We are studying
inherited
abnormalities of
sexual development
in dogs, focusing on
persistent Müllerian
duct syndrome and
XX sex reversal.*

Vicki Meyers-Wallen

Publications

The following is a list of manuscripts published by staff members of the Baker Institute in 1992. Publications listed as "in press" in last year's report are repeated this year, with their original numbers, to record their full bibliographic details.

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In the spring of 1993, Gustavo Aguirre will become the director of the Baker Institute, completing the process of transition that began two years ago. He will take charge of a dynamic and highly productive program of research that, though broad-based, continues to grow according to the dictates set down by Drew Baker and the founders of the Cornell Research Laboratory for Diseases of Dogs.

The preservation of that original mandate—to address the health needs of dogs in service to their human companions—has been one of Skip Carmichael's foremost concerns during the interim in which he has led the Institute. Skip answered the call out of a deep sense of responsibility to the Institute's mission and to its staff and contributors. During his brief stewardship, he has succeeded in setting a course for progressive change and the strengthening of existing disciplines that will endure and gain momentum under Gus Aguirre's leadership. He has done so with the support of an exceptional staff and a reinvigorated advisory council who share his dedication to the continued prosperity and prominence of the James A. Baker Institute for Animal Health.

The Institute has long flourished as a research center in an atmosphere of discovery and entrepreneurship within the College of Veterinary Medicine. Born in an era when infectious diseases, especially canine distemper, took a heavy toll, it has always had a strong connection with dog owners, kennel clubs, and veterinary practitioners. The generosity and influence of its backers have allowed Institute scientists to apply their expertise to emerging animal health issues with speed and notable effectiveness. We are pleased to see Dean Phemister's commitment to the principal of self-determination relating to the Institute's pursuit of its scientific mission. The Institute has earned its unique status many times over through its demonstrated capacity to muster the resources—both human and financial—to surmount problems of disease in animals.

The most recent example was the rapid development of means to control parvoviral disease in dogs and the exploration of how the newly emerged virus acquired the competence to infect and cause disease in dogs.

Gus Aguirre, a veterinarian and scientist, brings with him the scientific distinction, leadership qualities, and management experience needed to expand further the research potential of the Baker Institute. Those attributes, coupled with his genuine concern for companion animals, assure us that the Institute will continue to live up to its commitment to veterinarians, breeders, pet owners, the scientific community, and, above all, to dogs themselves.

*Robert E. Shope
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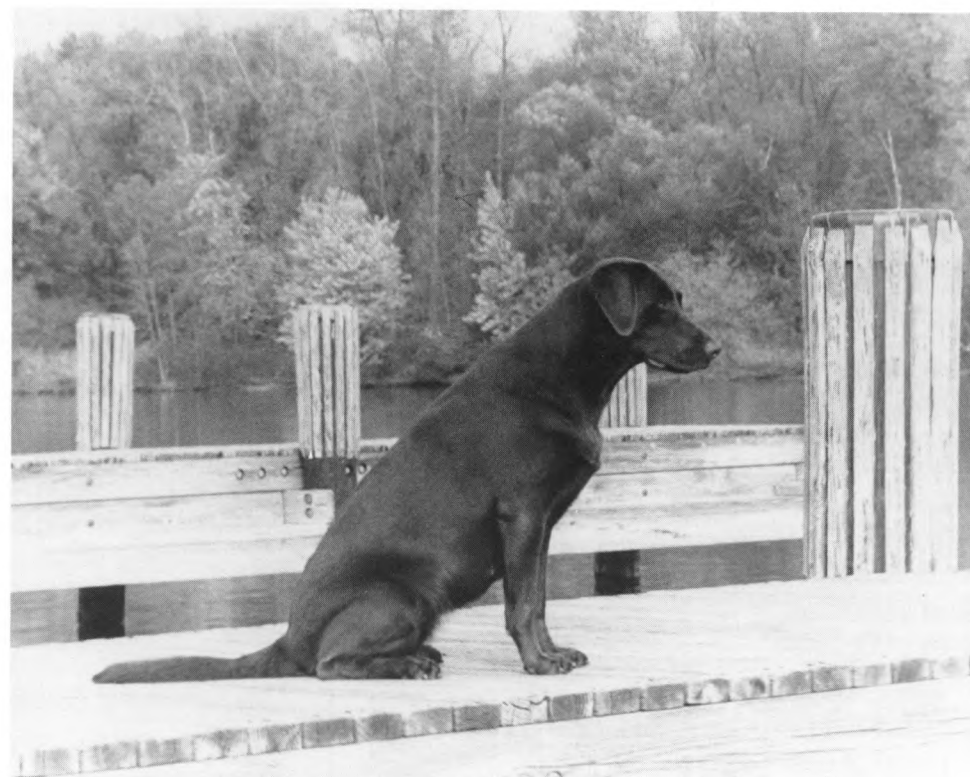
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*As a continuing memorial to Max, my Airedale, I
enclose my check for \$100.00 to assist in your
research in the diseases of dogs.*

*Max was my "live-in" family for more than 10
years. He was my constant companion—we climbed
Bluff Point hillsides, swam in Keuka Lake, sailed
together; we drove and flew together on trips; he was a
friend to the residents of the skilled nursing facility
where I worked—he was my best friend!*

*When out of love and consideration for him I
consented to his being euthanized, Eastview
Veterinary Clinic, who had always taken care of him,
sent a donation to you in his memory. It seemed such
a very fitting tribute I was moved to continue the
tribute.*

*My hope is that I may have helped to assure the
longer and healthier life of other equally loved dogs!*

*Frances T. Allison
Keuka Park, NY*

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... Doing something that extends the meaning of our pets' lives beyond the family circle and contributes to other pets not having to suffer from the diseases which claimed our own pets does help ease the loss of these special family members. We wish you well in your work.

*Patricia Trubow Hollister
Edgewood, KY*

- | | | | |
|--|--|--|--|
| Reverend Joseph D. Driskill
(in memory of Toby and
in honor of Dr. Charles J.
Berger) | Mr. and Mrs. Martin Elias
(in memory of Fu
Manchu) | Marianne M. Feraca
(in memory of Hans) | Mr. and Mrs. John A.
Frabotta
(in memory of Tilley) |
| Mr. and Mrs. Donald
Druding | Ellmers & Matochik | Bruce J. Ferguson
(in memory of Digger) | Paula M. Fraczek
(in memory of Sam) |
| Albert D. DuBell
(in honor of Roxy) | Elmwood Small Animal
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Ferguson
(in memory of Reda) | Kim Francisco |
| Daniel Duberman, DVM | Nancy A. Endres
(in honor of the marriage
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(in memory of Arthur) | Dr. and Mrs. William E.
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| Mr. and Mrs. Lee M. DuBois
(in memory of Benji) | Leona Epstein | Mr. and Mrs. Martin Feuer
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(in memory of Hippie) | Dr. and Mrs. Lincoln E.
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(in memory of Dawn) | Mr. and Mrs. William K.
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(in honor of Chipper) |
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(in memory of Poppy) | Christine M. Farley
(in memory of Grover) | Mr. and Mrs. Alan J. Finkel
(in memory of Taffy and
Snowball) | Norman T. Freid, DVM |
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(in memory of Hazy) | Mildred Farrell
(in memory of Ginger) | Patrice Fiorelli
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(in memory of Jounce) | Robert H. Featherston, DVM | Margot E. Fletcher | Mr. and Mrs. Gerald J.
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(in memory of Bear) |
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(in memory of Sparky and
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(in memory of Ranger) | Finita C. Fensel
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| | | Lorraine A. Fournier, DVM | Mr. and Mrs. Christopher P.
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| | | Mr. and Mrs. Thomas I. Fox
(in memory of Ginger and
Gretchen) | Daniel Garcia |
| | | Esta Fox
(in memory of Inga) | Garden State Animal
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| | | | Mr. and Mrs. George
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(in memory of Golden
and in honor of Dr. Bart
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Jim, and Andrew)

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(in memory of Puffy)

Sue Gehrlee
(in memory of Cyrus)

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Frances M. Gerdes

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(in memory of Mike)

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Mr. and Mrs. Jerry M.
Glassman
(in memory of Tiffany and
Stripsie)

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(in memory of Trixie and
Gilda)

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(in memory of Rosalind B.
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Martha Gold
(in memory of Dolly)

Mr. and Mrs. Herbert P.
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(in memory of Bubbles)

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Mr. and Mrs. Paul N.
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(in memory of Punkie)

Penny Gossweyler

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(in memory of Ginger,
Rachel, and Daisy)

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Margaret S. Gould
(in memory of Molly)

Lenore Grabow
(in memory of Spanky)

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(in memory of Puddy)

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(in memory of Eileen
Westfield and Tara)

Mr. and Mrs. Joe Graham
(in memory of Mitzi,
Shelley, and Patty Parrot)

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(in memory of Missy)

Nancy S. Graves
(in memory of Amy)

Mickey Henriques Gray

Lauren E. Green
(in memory of Jake and
Pepper)

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Hospital

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(in memory of Muffy and
Gavel)

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Adams Veterinary Clinic

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(in memory of Ebony)

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(in memory of Tisho, Teri,
Tobi, Ginger, Lita, and
Brandy)

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and Tracy)

Patricia Grove

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(in memory of Mindy)

Barbara G. Gubbins
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C. H. BROWN





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Mr. and Mrs. C. J. Guidroz
and family
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Marguerite B. Gulick, DVM

Jerome Gurewitz
(in honor of Dr. James
Jorgenson)

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(in memory of Dango)

Mary L. Guyette
(in memory of Diablo)

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(in memory of Tashi)

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Helen E. Hamilton

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Jeff Hanna
(in memory of Molly)

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(in memory of Nizam)

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(in memory of Kery)

Mr. and Mrs. James Haynie
(in memory of Shadow)

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Mr. and Mrs. Norman H.
Heaven
(in memory of Kenneth
Hammitt)

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(in honor of the marriage
of Dr. and Mrs. Kerry
Washburn)

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(in memory of Gunnery)

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Mallie, and Ike)

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Mr. and Mrs. Michael Hesser
and Amy
(in memory of Lassie)

Beth Hibbard
(in memory of Chester
and Brittany)

Elizabeth Hight

Mary Sue Hilliker
(in memory of
Arrowhead's Pink
Champagne)

Mr. and Mrs. Wendell Hilt
(in memory of Binker)

Klaus E. Hirmschal

Mr. and Mrs. John W.
Hirshfeld
(in memory of Mollie E.
Butler)

Mr. and Mrs. Robert
Hoagland
(in memory of Kodiak)

Jonathan A. Hoch

Jerry Hoffman
(in memory of Marian)

Ann F. Hoffman
(in memory of Hank)

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Hollander and family
(in memory of Max)

Mr. and Mrs. David M.
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(in memory of Misty)

Mr. and Mrs. Peter H.
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Paul J. Horohoe

Arthur M. Horowitz

Mr. and Mrs. Bruce F.
Horton and family
(in memory of Black
Magic)

Esther Hotaling
(in memory of Frank
Hotaling)

Drs. T. Richard and
Katherine A. Houpt
(in memory of Mollie E.
Butler)

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Janet C. Howard

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Michael Hubert

Hana Hudecek
(in memory of Buttons)

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Robert C. Hull (in memory of Butch)	Moelwyn Jones	Arlene W. Kettle	Mr. and Mrs. Stephen Kurlan and Liz (in memory of Libra and Shiloh)
Mr. and Mrs. Thomas H. Hunter	Mr. and Mrs. Matthew D. Jones (in memory of Mozee)	Kibbles'N Klips	Donald Kuropatwa (in memory of Shooter)
Mr. and Mrs. Daniel L. Hussin (in memory of Wink)	Mr. and Mrs. Anthony J. Joy, Jr.	Kenneth L. Kiehle, DVM	Mr. and Mrs. Robert J. LaTour (in memory of Scottie)
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Mr. and Mrs. Paul Imerese	Mr. and Mrs. Victor Jung (in memory of Alex)	Mr. and Mrs. Charles R. Kingsbury	Patricia Lafford (in memory of Crissie)
Dianna N. Indorf (in memory of Jake)	Mr. and Mrs. Ronald Junior (in memory of Frisca and Peppy)	Joan Barbara Kingsley (in memory of Leoa)	Florence R. Laicher (in memory of Ezra)
Kay Jackson (in memory of Muff)	Mr. and Mrs. Walter Justice (in memory of Webster)	Kinnelon Veterinary Hospital	Pamela Laidley (in memory of Pitter Patter)
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Dr. and Mrs. Stanley W. Jackson (in memory of Timothy, Mikhail, and Paddy)	Ida Marie F. Kahn	Beverly C. Klein	Mr. and Mrs. P. Henry Lambert
Jamie E. Jacobs (in honor of Robert A. Sparks and Ginger)	Abe B. Kamine, DVM	Mr. and Mrs. Robert Kline	Mr. and Mrs. Thomas W. Lamoreaux (in memory of Clancy)
Carl Jaeger	Mr. and Mrs. Bob Kane (in memory of Shannon)	Mr. and Mrs. David B. Klos (in memory of Aldomet)	Mr. and Mrs. Myron B. Lampert (in memory of Princess)
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Dr. and Mrs. Vernon H. Jensen (in memory of Mollie Butler)	Richard J. Kaufman	Dana Komjathy (in memory of Pebbles I and Bammer)	Cheryl G. Lappen (in memory of Mollie Butler)
Mr. and Mrs. Leo Jeyowski (in memory of Maxi)	Mary Lee Keating, DVM	Harold Kopit, DVM	Mr. and Mrs. Robert G. Lathrop (in memory of Archie)
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Christine L. Johnston, DVM	Mr. and Mrs. Steven C. Kellogg	Mr. and Mrs. David B. Kramer (in memory of Molly)	
Herbert Jonas, DVM	Amy Kelly	Roger E. Kranich (in memory of Schatzi, Taffy, and Tom)	
Cheryl A. Jones (in memory of Chaka)	Nada Kelly	Diane G. Kranz (in memory of Lukan)	
Caterina Jones (in memory of Shadow)	Robert L. Kennedy, DVM	Kathryn M. Kremer	
	Paul D. Kennett, DVM	Mr. and Mrs. Eric Krieger (in memory of Tiffany)	
	Robert E. Kesel (in memory of Pennie and Penney)	Mr. and Mrs. Richard Krongel	
	Joyce Kessel (in memory of General Lee)	Dr. and Mrs. Joseph Krug	
		Mr. and Mrs. Eric Kuhn	

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Mr. and Mrs. Sylvan Lazar	Pamela Lindner (in memory of Rapp and Lady)	Nan Macek-Rossignol (in memory of Shana)	Daniel J. Mazzocco
Robert K. Lee, DVM	Barbara C. Lippe (in memory of Poppins, Bubbie, Magus, and Samson)	Carolyn Mack	Patricia P. McCann (in memory of Penny)
Susan Leeds (in memory of Jenny, Samantha, and Juno)	Dr. and Mrs. Marc Lipton (in memory of Gwen)	Susan B. Mahar	Mr. and Mrs. Robert E. McCarty
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Mr. and Mrs. Ronald P. Lehrter	Joan LoFaso	Mr. and Mrs. David J. Mainville (in memory of Cyrus and Braun)	Frank E. McClelland, Jr., DVM §
Linda Leibfried (in memory of Princess)	Anne June Lohff (in honor of Tuffy and in memory of Trub, Wooly, Jet, Anthy, and Trygvie Lie)	Mr. and Mrs. Henry Majewski (in memory of Pride)	Douglas K. McClure
Carole M. Leichtung	Jean Lomba (in memory of Ginger)	Mr. and Mrs. Reino T. Makkonen (in memory of Dusty)	Anne McComb, DVM
Robert L. Leisten (in memory of Duke)	Helen B. Longo (in memory of Barbara Jacoby, Carmella Puccio, and Sandi Bluestein)	Mr. and Mrs. George A. Makrauer	Elizabeth Jane McCosh-Lilie
Carol Lenaghan	Robert A. Lopez, DVM	Linda Malanga (in memory of Brandi and Angel)	Ellen M. McCurdy
Mr. and Mrs. Robert D. Lenig	Karen Lorgeree	Nancy W. Maloney, DVM	Melanie McGarry (in memory of Ebony)
Winifred R. Leopoldt	Mr. and Mrs. Frederick Lossez (in memory of Natalie Grady)	Marilyn Mandelbaum	Mr. and Mrs. David A. McGoye (in memory of Max)
Richard F. Leskovar, DVM	Edward J. Loud	Leanna Manna (in memory of General Lee)	Marsha McKenna (in memory of Midnight)
Mr. and Mrs. Harry Lesseos	John Lucarelli (in memory of Sammy and Misty)	Robert V. Manning, DVM	Ursula McKinney (in memory of Daphne)
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Patricia W. Levin (in memory of Maggie)	Mr. and Mrs. Rein Lumi (in memory of Audrey, Scruffy, and Muffin)	Margot W. Marsh	Meadowview Kennels (in memory of Cody, Jada, Jaz, Charlie, Duchess, and Scheeka)
Ronnie Sue Levine	Mr. and Mrs. Irving M. Lustig	Patricia D. Martin (in memory of Annie Oakley and Blossom)	Flo Mehlman (in memory of Mollie Butler)
Myron Levinson (in memory of Freckles)		Mr. and Mrs. Raymond Matey (in memory of Baron Maximillian Von Blorg)	Rod Meier, DVM
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Brett Levitzke		Mr. and Mrs. Peter Mathews (in memory of J. D.)	
George Levy, DVM			
Mr. and Mrs. Hank Levy			
Mr. and Mrs. David Lewis (in memory of Jake)			
Evelyn Lewis (in memory of Tanya)			
Norman F. Lewis, DVM			

... You explained in your letter that Northside Animal Hospital sent Cornell Research Laboratory a thoughtful gift in memory of my beloved Ginger.

Time and again I read your letter, as it brings much comfort to me. I thank you for your very kind words.

At this time, I also would like to pay tribute to that beautiful, loving, faithful little friend that will forever remain in my heart.

Thank you, again.

*Dinah Oleszek
Bath, PA*

Donna Melliadis
(in memory of Rox-anne)

Rosa Mello

Jeanne Merich

Jane Merrill

Richard Merzon
(in memory of Dr. Erno
Hallo, Bingo, and Marie)

Metairie Small Animal
Hospital

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Mrs. Metzger
(in memory of Mollie E.
Butler)

Lynn D. Metzler

Frederick Meyerowitz
(in memory of Ginger and
Willy)

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(in memory of Bunny)

Mr. and Mrs. Peter J. Miller
(in memory of Sophia)

Mary Ann E. Miller

Barbara Miller
(in memory of Heather)

Andrew Miller

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(in memory of Gonzo)

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Mr. and Mrs. Craig F.
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(in memory of Bridgette)

Mr. and Mrs. Jack P.
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(in honor of Dr. Steven
Cohen and in memory of
Cinder)

Gary V. Montano
(in memory of Holly)

Lavonne Moody
(in memory of Cindy)

Bonnie Moran
(in memory of David
Moran)

Mr. and Mrs. Thomas D.
Morgan
(in memory of J. P.)

Christine M. Morris
(in memory of Bo-Bo)

Mr. and Mrs. Francis J.
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(in memory of Ryan)

Kathleen M. Morse
(in memory of Shanna,
Hobo, and Jasper)

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Mt. Rock Animal Hospital

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Munro, Jr.
(in memory of Coral)

Kathleen Murphy
(in memory of Roscoe)

Sean O. Murray

Alice Najarian
(in memory of Lassie,
Queenie, Kitty Kat, and
Spinner)

Nelson Najjum

Naomi Natcharian
(in memory of Rosalind B.
Moore)

Robert M. Naum, DVM

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(in memory of Missy and
Nicky)

Edith L. Nedwed

Madeline V. Nemeth

Pamela S. Nersesian, DVM

Constance B. Neustaetter
(in memory of Homer)

Brian Nevin
(in memory of Mollie E.
Butler)

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Hospital

Newport Veterinary Hospital

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Nicholas
(in memory of Samantha)

Josephine A. Nicholas

Allen E. Nichols

Christine M. Nicholson

Dorothy J. Nims
(in memory of Ralphie)

Normanside Veterinary
Clinic

Northside Animal Hospital,
Staten Island, NY

Norwichtown Veterinary
Hospital

Nottingham Pet Clinic

Mr. and Mrs. Charles Novak

Oakton Animal Clinic

Thomas M. O'Brien
(in memory of Ci)

Bob O'Bryan
(in memory of C. J.)

Mr. and Mrs. John
O'Connor
(in memory of Maya and
Oscar)

Lorrie Ann Ochman
(in memory of Isaiah)

Barbara M. Odell

Mr. and Mrs. John H.
O'Donnell
(in memory of Dr. Paul
Neussen and of Rudy)

Margaret Oettle
(in memory of Samson)

David J. Ohnemus
(in memory of Bubba)

Jean A. O'Keefe
(in memory of Rosie)

Old Lyme Veterinary Clinic

Dinah L. Oleszek
(in memory of Ginger)

Mr. and Mrs. Rick Olson

Mr. and Mrs. Frank L.
Oppenheimer
(in memory of Sammy)

Bernice Orlan
(in memory of Joshua)

Dr. and Mrs. Harold L.
Ornstein
(in memory of Polo)

Oronoque Animal Hospital

Mr. and Mrs. Sam Ortolano
(in memory of Shadow)

Carl Oshrain

Anna M. Ostrosky
(in memory of Tiko I and
Tiko II)

Herbert I. Ott, DVM

Theresa Ovitt
(in memory of Rosalind B.
Moore)

M. Josiah Owen and family

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Mr. and Mrs. Thomas Pall and family (in memory of Honey and Sasha)	Pamela S. Peters (in memory of Wendy)	Dennis W. Quirk	Mr. and Mrs. Daniel H. Riegler (in memory of Aslan)
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Johanna Paras (in memory of Dudley)	Mr. and Mrs. Joseph Petrillo (in memory of Dudley)	Anne Rajottes	Brian Rind, DVM
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Mr. and Mrs. Donald H. Parker (in memory of Blackie and Tippy)	Nancy L. Phaneuf (in memory of Mitzi)	Franklin W. Rapp, DVM	Susan G. Risberg (in memory of Bingo)
Belgica Pastrana	Phelps Veterinary Hospital	Judith A. Rapp	John F. Risickella (in memory of Joan Risickella and her dogs, Kim and Radar)
Mr. and Mrs. Perry S. Patterson (in memory of Linus)	Thomas J. Pierce	Jack D. Rasmusson, DVM	Sandra M. Risser (in memory of Taffy)
Mr. and Mrs. Stephen C. Paul (in memory of Chip)	Mr. and Mrs. John W. Pink (in memory of Jeffrey)	Virginia L. Ray (in memory of Dickens)	Rivergate Veterinary Clinic
Janka Pavelek (in memory of Hannah)	Marla A. Pinnataro	John J. Ray	Mr. and Mrs. Christopher Rivest (in memory of Ebony and Dusty)
Mr. and Mrs. Chester J. Pawenska	Aureo Pinto, Jr. (in memory of Jaws and Max Xerox)	Wilfred Raynor (in memory of Cleo)	Mr. and Mrs. John C. Rivosa (in memory of Chuck)
Elizabeth Pawlewski (in memory of Rosalind B. Moore)	Pioneer Valley Veterinary Hospital	Mr. and Mrs. Albert P. Raynor	Mr. and Mrs. Frederick Roberts (in memory of Bracken)
Paws-I-Tive Petcare (in memory of Willi)	Mrs. Collier Platt	Mr. and Mrs. Douglas K. Reading (in memory of Klaus and Jobie)	Elizabeth N. Roberts (in memory of Amber and Sean)
Jerome Payton, DVM	Pleasant Grooming: Steven Udell	Elizabeth Reardon (in memory of Sebbie)	Rocky Point Animal Hospital
Diana D. Peil (in memory of Sheila)	Michael L. Podolin, DVM	Lynnette Reede (in memory of Chillin and Pinky)	Dorothy G. Rodriguez
Sandra C. Pelesh (in memory of Sasha)	Patricia A. Pogue (in honor of Dr. Kevin Lynch and in memory of Willie)	Ida M. Reeder (in memory of Max and Brandy)	Mr. and Mrs. Robert N. Roeters
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Dr. and Mrs. Robert Perelman (in memory of Demian)	Potomac Animal Hospital	Catherine R. Reilly (in memory of Salty)	Phyllis A. Roitsch (in memory of High Sky's Concorde, C.D.X.)
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- | | | | |
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and Jason) |
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(in memory of Hank) | Marian E. Sage
(in memory of Penny) | Mr. and Mrs. John T. Scherff | Mr. and Mrs. Robert
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(in memory of Leif) |
| Robert Rosiek
(in honor of the marriage
of Dr. and Mrs. Kerry
Washburn) | Jo D. Samuels
(in memory of Diablo) | Mr. and Mrs. George J.
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Shelburne Veterinary Hospital			Mary Ann Taber (in memory of Ebony and in honor of Dr. David Stillman)
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Thekla Wacksman

B. Gertrude Wade
(in memory of Sahara)

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Rhonda Saltzman Wasserman

Waterbury-Oxford Veterinary Clinic

Samuel C. Weber
(in memory of Mrs. Mollie E. Butler)

Stanley A. Weber, Sr.

Lise Weil
(in memory of GiGi)

Inge I. Weil

West Hills Animal Hospital

West Roxbury Animal Hospital

Phyllis K. Wier
(in memory of Teddi)

Pamm G. Wiggins
(in memory of Shannon)

Charlene Wilcher

Dr. and Mrs. Arthur N. Wilder

... Truly, I wish the amount of the check could be in the millions, for the work that you are doing. I became aware of the Institute because of donations made in the names of two of my dogs by the Bolton Veterinary Hospital. I deeply cherish the thought behind those gifts, and I shall continue to tender whatever support I can.

*Doris C. Hattin
Bolton, CT*

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* Research Partner
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Airedale Terrier Club of Metropolitan New York (in honor of Dr. Alan S. Meyer)	Niagara Frontier Obedience Association * (in memory of Ch. Northeast's Sundance, Can. Ch. Northeast's Damien, Highgate's Boo De Bergler, Am/Can. Ch. Jassy Entre Deux Mondes TT H.I.C., and Atarot's Quick Silver CGC)
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Charles W. Allen
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Joan Risickella
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Anna Wollosinovich

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† We were saddened to learn
while preparing this report
of Dr. Fletcher's death on
New Year's Day of 1993.
Dr. Fletcher was the first
veterinarian to be
honored with the
Institute's Founders'
Award. His loss is greatly
felt.

Ways of Giving

In establishing the Baker Institute, of which the Cornell Research Laboratory for Diseases of Dogs is an important part, the Cornell University Board of Trustees authorized the Treasurer's Office of Cornell to be custodian of all funds given in support of the Institute. You are thus assured your gift will have the maximum benefit. There are many ways you can give to advance the work of the Baker Institute. Some of these opportunities offer substantial income tax and estate tax benefits.

Checks

All checks should be made payable to the Baker Institute and mailed to:

**Office of the Director
James A. Baker Institute for Animal
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Cornell University
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Appreciated Stocks

Selling appreciated stocks is almost certain to increase your taxes, but if you give the stocks to Cornell outright and deduct their full current market value as a charitable contribution, you can probably avoid all or most of the capital gains tax. To complete the transaction with maximum speed and at lowest cost,

1. take the certificate to your bank or broker;
2. inform your bank or broker that you want to make a gift of the shares or securities to Cornell University for the Baker Institute;
3. instruct your bank or broker to telephone the **Office of Trusts and Estates at 607 277-0025**; and
4. write a note to the Director, James A. Baker Institute for Animal Health, Cornell University, Ithaca, New York 14853, including the name of your bank or broker and the form and size of your gift.

Depreciated Stocks

You get a maximum benefit from a gift of depreciated stocks by selling them and giving the cash to Cornell. That way you get the capital loss allowance and a charitable contribution deduction for the amount of the gift. Instruct your bank or broker to sell the shares or securities and send the proceeds as a gift to Cornell for the James A. Baker Institute for Animal Health.

Bequests

Charitable bequests provide substantial estate tax benefits. They can be gifts of land or buildings, securities, personal property, or cash. The Cornell University counsel suggests the following provision: **I hereby give, devise, and bequeath [description of property] to Cornell University, an educational corporation located at Ithaca, New York, for the uses and purposes of the James A. Baker Institute for Animal Health.**

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An income-producing trust enables you to make a gift to the Baker Institute, gain income for life, and derive tax benefits. A beneficiary may be named to receive the income, too. The Institute offers three plans: the **Pooled Life Income Fund**, the **Annuity Trust**, and the **Unitrust**. You may also establish



a **Charitable Lead Trust**, which will first pay income to Cornell and later transfer assets back to you or a third party. Financial planning involving deferred gifts requires expert advice from your attorney and other specialists. If you are interested in deferred giving, please notify the director, who will make arrangements for you to receive more information.



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FOR
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DEDICATED 1975**